COMPARISON OF HEMODYNAMIC EFFECTS OF LIGNOCAINE 2% WITH ADRENALINE IN TWO DIFFERENT CONCENTRATIONS OF 1:80,000 AND 1:2, 00,000

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CERTIFICATE

This is to certify that this dissertation titled "**COMPARISON OF HEMODYNAMIC EFFECTS OF LIGNOCAINE 2% WITH ADRENALINE IN TWO DIFFERENT CONCENTRATIONS OF 1:80,000 AND 1:2, 00,000**", is a bonafide record of the work done by **DR.NANDAGOPAN S** under my guidance during his post graduate study period from 2010 to 2013 under THE TAMIL NADU Dr.MGR MEDICAL UNIVERSITY, CHENNAI, in partial fulfillment for the degree of **MASTER OF DENTAL SURGERY IN ORAL AND MAXILLOFACIAL SURGERY, BRANCHIII**. It has not been submitted (partial or full) for the award of any other degree or diploma.

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CONTENTS

LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
ASA	American Society of Anesthiologists
BP	Blood Pressure
DBP	Diastolic Blood Pressure
HR	Heart Rate
LA	Local Anesthesia
min.	Minute
ml	Milli liter
mg	Milli gram
pg	Pico Gram
SBP	Systolic Blood Pressure
SD	Standard Deviation
SPO_2	Oxygen Saturation
SPSS	Statistical package for social sciences

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ABSTRACT

COMPARISON OF HEMODYNAMIC EFFECTS OF LIGNOCAINE 2% WITH ADRENALINE IN TWO DIFFERENT CONCENTRATIONS OF 1:80,000 AND 1:2,00,000

Aims & Objectives:

The present study was designed to compare the hemodynamic parameters during administration of local anesthetics agent lignocaine 2% without adrenaline and with adrenaline in different concentrations such as 1:80,000 and 1:2, 00,000 in minor oral surgical procedures with regard to systolic blood pressure, diastolic blood pressure, heart rate and oxygen saturation

Materials and Methods

Thirty ASA grade I patients were selected who were to undergo multiple minor oral surgical procedures. All patients received 1.8 ml of local anesthetic solutions during each appointment. All patients were categorized into groups based on the choice of local anesthetic agent used with there respective concentration of adrenaline. The total no. of groups being three, group I included patients who received plain Lignocaine 2%, group II received Lignocaine 2% with adrenaline 1:80,000 and group III received Lignocaine 2% with adrenaline 1:2, 00,000. Under each group, the following parameters such as oxygen saturation, heart rate, systolic and diastolic blood pressure were recorded immediately after administration and at timely intervals of 5 min, 10 min, 15 min, 20 min and 30 min.

Results:

It was noticed that oxygen saturation did not show any variations while using lignocaine 2% or lignocaine 2% with adrenaline 1:80,000 or lignocaine 2% with adrenaline 1: 2,00,000. Those patients who received plain lignocaine 2% showed only minor variations in HR which was not significant while those patients who received lignocaine 2% with adrenaline 1:80,000 and lignocaine 2% with adrenaline 1:2,00,000 showed an increase in heart rate after administration of local anesthetic. Patients receiving local anesthetic with adrenaline in concentrations of 1:80,000 and 1:2,00,000 showed a bimodal change in systolic blood pressure. An initial decrease in systolic blood pressure was noted immediately after administration and then increases at 10 min following administration. Again after 10 min period, systolic pressure decreased gradually till 30 min. Diastolic blood pressure did not show any variations in those patients who received plain lignocaine 2% but was noted to be decreasing in patients receiving local anesthesia with adrenaline in concentrations of 1:80,000 and 1:2,00,000.

Conclusion:

To conclude, even though use of lignocaine with adrenaline is associated with changes in blood pressure and heart rate, clinically they are less significant and such changes did not produce any marked systemic untoward outcome at any of the studied adrenaline concentrations of 1:80,000 or 1:2,00,000.

Vasoconstrictors are those drugs that constrict blood vessels and thereby controlling tissue perfusion. Most local anaesthetics, except cocaine, are vasodilators. Vasoconstrictors are added to local anaesthetic agents to counter their vasodialatory effects.

Vasoconstrictors used commonly in conjunction with local anaesthetics are chemically similar to sympathetic nervous system mediators – epinephrine and norepinephrine. So their actions resemble adrenergic nerve stimulation. These drugs being classified as sympathomimetic or adrenergic drugs have several actions besides vasoconstriction.

The systemic actions of the sympathomimetic amines can be classified into five broad types. (1) A cardiac excitatory action resulting in an increase in heart rate, force of contraction and stroke volume. (2) A central nervous system excitation resulting from the vasoconstrictor agents. (3) Metabolic actions, such as increase in the rate of glycogenolysis in the liver. (4) Peripheral excitatory action on smooth muscles, including those in blood vessels supplying mucous membrane and skin, providing vasoconstrictor effect. (5)The sympathomimetic effect may cause a peripheral inhibitory action on certain other types of smooth muscle, such as those in the bronchial tree and in the gut wall[1].

The clinical efficiency of a local anaesthetic agent is dependent upon the action of the vasoconstrictors. Vasoconstrictors added to local anaesthetic solution provide several advantages to the anaesthetic. First, the addition of vasoconstrictor increases the duration and depth of anaesthesia and pain control. Second, it can decrease the systemic toxicity of local anaesthetics by retarding their absorption into

the systemic circulation. Third, the vasoconstrictor provides haemostasis at the surgical site and reducing blood loss. Finally, the addition of vasoconstrictor can enhance the quality of the neural blockade.[2]

Epinephrine is the most commonly employed vasoconstrictor in dentistry. As with the other useful vasoconstrictors, epinephrine produces its effects by stimulating the alpha adrenergic receptors located in the walls of the arteriole. Epinephrine is also a beta adrenergic stimulator and may cause vasodilatation of arterioles in skeletal muscle due to the predominance of beta receptors in this tissue. Epinephrine's beta adrenergic responses, even at low systemic levels, include skeletal muscle vasodilatation with increased heart rate. The beta adrenergic effects predominate over the alpha because of the greater sensitivity of beta adrenergic receptors to epinephrine.

The technique of diluting vasoconstrictors in local anaesthetics dates back to 1897 when Abel discovered adrenaline. Vasoconstrictors commonly used in dental surgery are of various dilutions of 1: 80,000, 1: 1, 00,000 and 1: 2, 00,000 having 0.0125 mg/ml, 0.01 mg/ml and 0.005 mg/ml of epinephrine respectively.

During local anaesthetic administration, epinephrine is absorbed from the injection site just as the local anaesthetic which can influence the heart and blood vessels. Lignocaine with adrenaline 1: 2, 00,000 dilution on administration can double the plasma epinephrine concentration[3].There has long been debate regarding the safety of using epinephrine as a vasoconstrictor with local anaesthetics. Current evidences in contrast to previous studies demonstrates that intra oral injection of local anaesthetics can elevate the plasma epinephrine concentration equivalent to that achieved during moderate to heavy exercise[4]. This is associated with moderate

increase in blood pressure and heart rate. Intra vascular administration of epinephrine 0.015mg can cause an elevation of heart rate of 25 – 70 per minute and systolic blood pressure of 20 -70 mm Hg.

Inadvertent intravascular administration, injections of increased volumes or concentrations, or injection into inflamed tissue may enhance the systemic uptake of vasoconstrictor and local anaesthetic and produce toxic manifestations. The signs and symptoms of vasoconstrictor over dosage include tachycardia, hypertension, tremors, headache, palpitations and, rarely ventricular fibrillation by direct effect on the myocardium.

The effect of local anaesthetics with adrenaline in terms of potency and its action is well established and documented[5, 6]. However hemodynamic parameters with respect to systemic response need further investigations and understanding. Changes such as variable blood pressure, peripheral pulse and heart rate during administration and its effect till the cessation of procedure lack clarity. With respect to local anaesthesia with different concentrations on adrenaline as well as without adrenaline producing such changes require further evaluation based on their systemic effects and outcome.

The present study was designed to assess and compare the hemodynamic effects of 2% lignocaine with adrenaline in concentrations of 1: 80000 & 1: 2, 00,000 during routine dental extractions in normal healthy individuals.

AIMS

The aim of this study was to quantitatively assess different hemodynamic parameters during minor oral surgical procedures under local anaesthesia without the use of adrenaline and with adrenaline at different concentrations of 1:80,000 and 1:2,00,000.

OBJECTIVES

The present study was designed to compare the hemodynamic parameters during administration of local anaesthetics agent lignocaine 2% without adrenaline and with adrenaline in different concentrations such as 1:80,000 and 1:2, 00,000 in minor oral surgical procedures with regard to:

1. Systolic blood pressure.
2. Diastolic blood pressure.
3. Heart rate.
4. Oxygen saturation.

Vernale et al[7] in 1962 investigated the cardiovascular effects of injection of 2% lignocaine with and without epinephrine 1:100,000 in normotensive and hypertensive subjects. This study revealed that the cardiovascular responses of the hypertensive patients differed from that of the normal subjects. A rise in the systolic pressure and heart rate and a slight rise or fall in the diastolic pressure was noticed in normotensive and hypertensive group. Hypertensive patients reacted more vigorously to pressor stimuli. He concluded that no significant variations in cardiovascular responses in blood pressure, heart rate, and ECG complex were produced by injection or extraction in either the control or the epinephrine treated groups of both normotensive and hypertensive patients.

Stannard et al[8] in 1968 evaluated the hemodynamic effects of lignocaine during acute myocardial infarction. A 100 mg of lignocaine was administered over a period of 5 min in patients with myocardial infarction within 24 hrs. No statistically significant change in blood pressure was seen before and after administration of lignocaine. Hence they concluded that lignocaine is a safe drug to be used in ventricular arrhythmias complicating acute myocardial infarction.

Christensen et al[9] in 1980 measured blood pressure intra-arterially after subcutaneous infiltration of 15-20 ml of 0.5% lignocaine with nor adrenaline (1microgram/ml) in 20 patients aged 24-73 who were scheduled for craniotomy. In this study SBP went up to 125% of the pre injection values, reaching a maximum seven minutes after injection. Within 20 minutes, the blood pressures fell to 105% of pre injection values. They concluded that use of adrenaline should be considered an additional hazard to patients undergoing neurosurgical operations.

Goldstein et al[10] in 1982 studied the circulatory, physiological and plasma catecholamine responses during third molar extractions and the effect of sedation with intravenous diazepam and inclusion of epinephrine in local anaesthetic. The HR and SBP increased significantly in all patients and no change in DBP was seen. Non sedated patients showed a 60% rise in plasma nor epinephrine concentration. Administration of local anaesthetic containing epinephrine resulted in a rise in mean plasma epinephrine by 5 minutes after injection. The results suggested the involvement of sympathetic nervous system in producing circulatory responses in dental surgery.

Chernow et al[11] (1983) conducted a randomized double-blind crossover trial to define the hemodynamic effects of local anaesthesia during dental procedures. The mean arterial pressure (MAP), heart rate, and plasma catecholamine responses for 60 minutes following an inferior alveolar nerve block with epinephrine and norepinephrine-containing lignocaine hydrochloride in 14 men were measured. The results showed that lignocaine alone caused no significant change in mean arterial pressure or heart rate and only slight, transient changes in plasma catecholamine concentrations when compared with baseline values. Lignocaine with epinephrine caused significant, sustained increases in plasma epinephrine concentrations and a slight, but transient increase in heart rate from 68 to 70 beats per minute. Lignocaine with epinephrine caused no significant change in MAP. They concluded that there is no significant hemodynamic response to lignocaine dental anaesthesia with or without epinephrine in healthy young men.

Emanuel S. Troullos, (1986)[12] evaluated the effects of administering an epinephrine-containing local anaesthetic on plasma catecholamine levels and

cardiovascular parameters. Significant elevations were observed following administration of 8 dental cartridges of 2% lignocaine with epinephrine 1:100,000 (144 µg). Minimal changes were observed in the patients who received 6 cartridges of 3% mepivicaine. One minute after injection, the mean plasma epinephrine level in the group receiving epinephrine was 27 times higher than baseline. There was significant elevation in systolic blood pressure and pulse rate with the epinephrine group. No significant change in diastolic pressure was seen. These results indicate that significant amounts of epinephrine can be systemically absorbed following intraoral injection and the absorbed epinephrine can alter the cardiovascular status of the patient.

Meyer F U[13] in 1986 conducted a prospective randomised study of hemodynamic changes of normotensive and hypertensive groups undergoing extraction procedures under 2% lignocaine with and without 1:100,000 epinephrine. No significant differences were noticed between the groups in evaluated hemodynamic parameters. He reported that in both groups the changes of blood pressure and heart rate were similar.

Abraham[14] in 1988 studied changes in the cardiovascular system before, during, and after treatment for 40 patients undergoing extractions. He found that patients with hypertension undergoing dental extractions had a greater increase in blood pressure than normotensive patients after injection of 2% lignocaine with 1:80,000 epinephrine. In addition, 7.5% of the patients with hypertension developed significant arrhythmias.

Salonen et al[15] in 1988 conducted a double-blind, cross-over study to evaluate the role of adrenaline 1:80,000 in lignocaine used in dental local anaesthesia on

hemodynamics and plasma catecholamine levels. The exogenous adrenaline significantly elevated the heart rate, but did not affect systolic or diastolic blood pressure. They conclude that the adrenaline present in the local anaesthetic is a major source of adrenergic activation during minor oral surgery.

Niwa et al[1] in 1991 compared the effects of infiltration injection of 3.6 mL lignocaine with either 1:80,000 epinephrine or 1:25 norepinephrine. It was concluded that epinephrine activates left ventricular diastolic function and in contrast norepinephrine impairs it.

Meechan et al[16] in 1992 studied metabolic responses to oral surgery under local anaesthesia and sedation with two different local anaesthetics. The patients were randomly given 4.4 mL of 2% lignocaine with 1: 80,000 epinephrine in one group and 4.4 mL of 3% prilocaine with 0.03 IU/mL felypressin in other group. The epinephrine-containing local anaesthetic reduced the plasma potassium concentration at 10 min after injection and increased the blood glucose concentration. Plasma potassium increased and blood glucose decreased following the administration of the epinephrine free solution. So they concluded that epinephrine-free and epinephrine containing local anaesthetics differs in their metabolic effects during oral surgery.

Peruse et al[17] in 1992 did a literature review regarding the contraindications to vasoconstrictors in dentistry. It was suggested that the absolute contraindications include heart diseases such as unstable angina, recent myocardial infarction, recent coronary artery bypass surgery, uncontrolled hypertension, diabetics, hyperthyroidism, sulphite sensitivity and pheochromocytoma. The relative contra indications include patients under tricyclic anti depressants, phenothiazine compounds, monoamine oxidase inhibitors, non-selective beta blockers and cocaine

abusers. They concluded that complications occurring after administration of local anaesthetics with vasoconstrictors are not exclusive for patients with severe cardiovascular disease.

Middlehurst [18] in 1999 investigated hemodynamic and electrocardiographic responses to lignocaine 2%, noradrenalin 1:50,000, vasopressin 0.25 IU/mL, and midazolam. There was statistically significant changes in the individual parameters of mean heart rate and mean systolic blood pressure with the administration of anaesthetic. The values were rising from a baseline value to a peak and then falling. But on considering statistically significant changes to the mean values for HR and SBP for both anaesthetic and sedation groups, the physiological magnitude and clinical relevance of such change would be described as minimal.

Homma et al[19] in 1999 studied the relationship between oral mucosal blood flow and plasma epinephrine levels after sub mucosal epinephrine injections. Patients were administered with 2% lignocaine with adrenaline submucosally. Plasma epinephrine concentration, SBP, DBP and HR were recorded before and after infiltration. There was an increase in plasma epinephrine concentration which peaked by 3min after infiltration. A correlation was present between base line mucosal blood flow and maximum increase in plasma epinephrine concentration. There were no significant differences in the hemodynamic changes. This result suggests that plasma epinephrine concentration after sub mucosal infiltration is dependent on the initial mucosal blood flow in the injected areas.

Yagiela et al [20] in 1999 reviewed the literature on drug interactions associated with vasoconstrictors in dental practice involving tricyclic antidepressants, nonselective β-adrenergic blocking drugs, certain general anaesthetics and cocaine.

Tricyclic antidepressants block muscarinic and α1-adrenergic receptors and directly depress the myocardium. These actions can additionally modify cardiovascular responses to the vasoconstrictors. Use of epinephrine in halothane anaesthesia was associated with ventricular dysrythmias. In case of cocaine abusers, cocaine may enhance adrenergic neurotransmitter release and postsynaptic responses are intensified to epinephrine like drugs. Hence vasoconstrictors should be with held for at least 24 hours after cocaine exposure to allow for elimination of the drug and its active metabolites. In conclusion, careful administration of small doses of vasoconstrictors, coupled with monitoring of vital signs, will permit these drugs to be used with only minimal risk.

Niwa et al[21] in 2000 investigated the hemodynamic response to infiltration anaesthesia and epinephrine infusion and compared these responses with those produced by ergo meter exercise. The study subjects were given epinephrine infusions to produce cardiovascular response comparable to those seen in lignocaine – adrenaline (1:80,000) infiltration anaesthesia. The cardiovascular response to this drug was evaluated using echocardiography and compared to those produced by ergo meter exercise. The results revealed that the cardiovascular response to infiltration anaesthesia is minimal and is equivalent to walking at a speed of 4.8 km/hr. So it was concluded that except in certain pathologic conditions such as serious arrhythmia, infiltration anaesthesia can be carried out safely.

Tanaka et al[22] in 2000 evaluated the efficacy of hemodynamic criteria for detecting intravascular injection of epinephrine. Patients under general anaesthesia were given epinephrine test dose of 15 micro grams. HR, BP and ECG were recorded before and after administration of test dose. Compared to pre-injection values,

injection of epinephrine test dose produced significant increase in HR between 40–80seconds. A significant decrease in HR was seen between 160–280seconds. A significant, monophasic increase in SBP were seen between 20–240seconds. These results indicated that a minimal effective epinephrine dose for detecting unintentional intravascular injection of epinephrine was 15 micrograms creating > 10 beats/min increase in HR and the SBP > 15 mmHg than baseline values.

Meechan et al [23] in 2001 performed a randomised, single-blind, cross-over study to evaluate the hemodynamic effects of 2% lignocaine with 1:80,000 epinephrine and 3% prilocaine with felypressin. There was an increase in heart rate 10 minutes following the injection of the epinephrine-containing solution and fall in diastolic blood pressure 20 minutes after injection of lignocaine with epinephrine.

Niwa H et al[24] in 2001 conducted a study to examine the safety of epinephrine-containing local anaesthesia for use on patients having cardiovascular disease with impedance cardiology to determine hemodynamic responses to an injection of 1.8 mL of 2% lignocaine with 1:80,000 epinephrine. The conclusion was that lignocaine-epinephrine was safe and had few, if any, hemodynamic consequences in patients with cardiovascular disease.

Fernieini M et al[25] in 2001 compared the hemodynamic effects of local anaesthetics with or without adrenaline using laser doppler flowmetry. The heart rate increased in plain lignocaine and lignocaine with adrenaline group just before administration and returned to normal immediately after injection. Again a significant increase in HR was seen in epinephrine group 5min post injection and persisted to 10 min post injection with the epinephrine containing LA in this study. The author suggested the pre injection rise in HR was a result of endogenous release of

catecholamine. The delayed increase in the HR in adrenaline group was due to adrenaline contained in local anaesthetics.

Niwa et al[26] in 2001 evaluated hemodynamic responses to intraoral injection of lignocaine with 1:80 000 adrenaline with impedance cardiograph to examine their safety on patients with cardio vascular disorders. In this study the SBP and HR was increased by 4.1% and 5.1%. DBP remained unchanged. None of the patients had any cardiac symptoms and no significant response related to the extent of cardiac functional capacity. They concluded that lignocaine epinephrine was safe with minimal hemodynamic consequences in patients with cardio vascular disorders.

Ohkado et al[27] in 2001 compared the anaesthetic potency of lignocaine and epinephrine in different concentrations. Anaesthetic potency on dental pulp was investigated by changing the concentration of lignocaine and that of epinephrine. The results showed that a decrease in the epinephrine concentration produced a decrease in the anaesthetic potency independent of lignocaine concentration. It was concluded that an increase in lignocaine concentration may not compensate the decrease in epinephrine concentration.

Murthy and Rao[28] in 2001 investigated cardiovascular responses to infiltration of the scalp with five different combinations of epinephrine and lignocaine. Group A being lignocaine 0.5%; Group B- lignocaine 0.5% with epinephrine 1:200,000; Group C - lignocaine 0.5% with epinephrine 1:100,000; Group D- normal saline with epinephrine 1:200,000; and Group E- normal saline with epinephrine 1:100,000. Plain lignocaine did not cause any significant change in blood pressure. The systolic, diastolic, and mean arterial hypertension was significantly increased in group E. Episodes of diastolic hypertension occurred frequently in Group D. A biphasic

diastolic and mean arterial hypotension by around minute 2 and minutes 9-15 occurred in both Groups C and B. In conclusion, epinephrine in concentrations of 1:100,000 and 1:200,000 causes significant tachycardia and hypertension. The combination of lignocaine and epinephrine attenuates the hypertension but results in a biphasic hypotensive response.

Bader et al[29] in 2002 conducted a review of cardiovascular effects of epinephrine on hypertensive dental patients. The results revealed that epinephrine use in hypertensive patients is associated with increase in SBP for 4 mmHg and no higher for normotensive subjects. HR was higher in patients receiving epinephrine than that with plain lignocaine group and DBP was found to decrease for both normotensive and hypertensive individuals.

Faraco et al[6] in 2003 conducted a double blind study to evaluate cardiovascular parameters during dental procedures. The parameters included SBP, DBP, and mean blood pressures, and heart rate. The patients were divided into 3 groups. One group received no premedication. Group 2 with 10 mg diazepam, and group 3 premedicated by a placebo. All patients received 1.8 mL of 2% lignocaine with epinephrine 1:100,000. There were no changes in the parameters evaluated during the clinical procedures. They concluded that there is no change in the cardiovascular parameters with pre medication during dental procedures.

Yang JJ et al[30] carried out a prospective, randomized; double-blinded study to discover the hemodynamic effects after local infiltration of 1:200,000 adrenaline contained in 2% lignocaine under general anaesthesia. It revealed Lignocaine 2% or saline with adrenaline (1:200,000) does cause temporary hypotension and other hemodynamic changes during general anaesthesia, which last no longer than 4

minutes. This mechanism was attributed to the preferential stimulation of the beta 2 receptors at lower concentrations of adrenaline.

Gedik et al[31] in 2005 investigated changes in blood pressure, pulse rate and temperature before and after periodontal surgery. A decrease in BP, HR and temperature were observed during administration of local anaesthetic containing 0.06mg adrenaline. They concluded that factors such as patient age, gender, volume of local anaesthetic, the length of the treatment and the difficulty of the procedure may be determinants of the change in blood pressure. All the parameters that showed significant changes only in medically compromised patients.

Meral et al[32] in 2005 conducted a randomised cross over comparison to determine the effects of lignocaine with or without adrenaline on plasma epinephrine concentrations and hemodynamic changes during third molar surgery. The results revealed there is no significant change in the SBP or DBP or saturation in both groups. The plasma epinephrine values were higher in lignocaine with adrenaline group. He concluded that local anaesthetic with adrenaline has minimal hemodynamic consequences on third molar surgeries.

Haghighat et al[33] in 2006 evaluated hemodynamic effects of 2% lignocaine with 1:80,000 epinephrine in inferior alveolar nerve block. Systolic blood pressure was raised during the injection, but 10 minutes later, it was lower than that during injection. A decrease in diastolic blood pressure was noticed at the end of injection and 10 minutes after injection. He suggested that the increase of BP at the beginning of dental operation was probably due to stress. Considerable decrease in diastolic BP after injection could be because of decrease in dental syringe phobia after local anaesthesia infiltration. Findings of this study showed that one cartridge of lignocaine

with epinephrine has minimal clinical effects on BP, and the prescribed dosage is without any severe hemodynamic effect on healthy patient.

Niwa et al[34] 2006 studied the cardiovascular effects of epinephrine under sedation with nitrous oxide, propofol, or midazolam. Hemodynamic response and blood pressure and heart rate were measured. When epinephrine was infused alone, heart rate (HR) and cardiac index (CI) increased. Propofol suppressed the epinephrine-induced increase in CI. In case of midazolam infusion, the highest dose of adrenaline caused a significant increase in HR, which was significantly higher than for epinephrine infusion alone. Nitrous oxide has no influence on the cardiovascular response to epinephrine. They concluded that increased cardiovascular activity due to epinephrine can be alleviated by propofol.

Yang et al[35] in 2006 conducted a prospective randomized double blind study to observe hemodynamic changes caused by local infiltration of epinephrine containing lignocaine solution on nasal field under GA. This study showed local infiltration of epinephrine-containing lignocaine solution on nasal field causes significant decrease in mean arterial pressure and increase in HR. The explanation was the hemodynamic effects of epinephrine are dose-dependent and different dose of epinephrine may activate different types of sympathetic receptors. A rate of 1 to 2μg/min predominantly activates β2-receptors with resulting vascular and bronchial smooth muscle relaxation and a rate of 2 to 10 μg/min predominantly activate β1-receptors to increase heart rate, contractility, conduction and decrease the refractory period. Doses in excess of 10 μg/min cause marked α-stimulation with resultant generalized vasoconstriction. The hemodynamic changes after local infiltration of

epinephrine depend on physical status of the individual, epinephrine dose used, vascularity of the site of administration and its rate of absorption from infiltrated area.

Faraco et al[36] in 2007 conducted a study to evaluate cardiovascular changes during dental implant surgery. It was done using 2% lignocaine with 1:80,000 epinephrine. The patients were monitored in the preoperative, intraoperative, and postsurgical periods by continuous non-invasive automatic arterial pressure and heart rate measurements taken every 2 minutes. No significant change in the evaluated parameters was noticed. They concluded that there were no changes in the analyzed cardio circulatory parameters during dental implant surgery in relation to systolic, diastolic, and mean arterial blood pressures and heart rate in normotensive subjects anesthetized with 2% lignocaine with epinephrine 1:80,000.

Liau et al[37] in 2008 evaluated the cardiovascular response to dental anxiety during local anaesthesia for dental procedures. BP, HR, SPO_2 and ECG changes were measured before and after the administration of anaesthetic. Anxiety was measured 15 minute before the procedure using Corah's Dental anxiety scale. Patients with mild dental anxiety had lower HR than those with moderate or severe dental anxiety. After local anaesthetic administration, HR increased in all patients and the increase was greater in severe dental anxiety group. The increase in SBP was greater in severe dental anxiety group than others. No significant change in oxygen saturation was noticed. This suggests that dental anxiety impacts the effect of delivery of local anaesthesia on BP and heart rate.

Ricardo et al[38] in 2008 conducted a double-blind randomized clinical trial to examine the effect of four different local anaesthetics of the amide group in patients undergoing extraction of lower third molars and verified the changes in systolic,

diastolic and mean blood pressures, heart rate (HR) and oxygen saturation (SPO_2). It was seen that all the anaesthetics produced an increase in HR. The variations in BP, HR and SPO2 returned to normal without the need for any additional treatment. So they concluded that local anaesthetics, when used in dentistry, produce limited and safe hemodynamic effects.

Moshaver et al[39] in 2009 conducted a double-blind, randomized clinical trial to assess the hemodynamic and haemostatic effects of 2 different concentrations of epinephrine in local anaesthetic used during functional endoscopic sinus surgery. Baseline and post injection hemodynamic parameters were recorded at 1-minute intervals for 5 minutes. Hemodynamic and haemostatic parameters and intraoperative blood loss were compared. Significant hemodynamic fluctuations were noted following injection of lignocaine, 2%, with 1:100 000 epinephrine. Increase in heart rate and systolic, diastolic, and mean arterial blood pressure were noted in 1:100 000 epinephrine group. The increase was found to be significant in the first and second minutes after injection and decreased to baseline level by the fifth minute. This fluctuation was not noted in patients, who received lignocaine, 2%, with 1:200000 epinephrine.

Laragnoit et al[40] in 2009 conducted a randomised clinical trial to analysed the hemodynamic changes in patients with cardiac diseases undergoing dental treatment under local anaesthesia with 2% lignocaine and adrenaline 1:2.00.000. Patients were divided into two groups. One group received plain lignocaine and the other group received lignocaine with adrenaline. No difference in SBP, DBP and HR were noticed before and after administration of local anaesthetic between the two groups. They

concluded that administration of local anaesthetic with adrenaline in cardiac valvular disease patients did not cause any arrhythmic or cardiovascular changes.

Marcelo et al[41] in 2010 evaluated glucose levels and hemodynamic changes in patients during routine dental treatment with and without local anaesthesia. Hemodynamic parameters, including SBP, DBP, HR, and glucose levels did not change significantly regardless of whether local anaesthesia was used. He explained the reason for no change in hemodynamic parameters that adrenaline has both beta 1 and beta 2 activity. Beta 1 stimulation causes an increase in blood pressure, whereas beta 2 stimulation decrease blood pressure.

Tariq et al[42] in 2010 conducted a comparative study to find out changes in the systolic blood pressure, heart rate, and diastolic blood pressure after infiltrating scalp with adrenaline with or without Lignocaine. One group received lignocaine 2% with adrenaline 1:200,000 and group 2 received normal saline with adrenaline 1:200,000. Tachycardia was seen more frequently in second group. SBP, DBP and mean arterial pressure were significantly increased in group 2. Significant decrease in DBP occurred in group 1. This showed that scalp infiltration of adrenaline alone will cause significant hypertension. The addition of lignocaine decrease in blood pressure.

Goranovic et al[43] in 2011 conducted a retrospective, comparative, non-randomised, study to determine the effect of injection speed of adrenaline- containing lignocaine solution on hemodynamic changes during local infiltration of nasal mucosa under general anaesthesia. There was no significant difference in HR, SAP, DAP nor MAP during either slow (< 60seconds) or fast (> 60seconds) injections. However lignocaine with adrenaline induced a decrease of blood pressure.

Vnuk et al[44] in 2011 evaluated hemodynamic effects of epidural lignocaine and lignocaine-adrenaline in dogs. It was noticed that heart rate and cardiac output increased significantly in adrenaline compared to baseline values. There was a decrease in arterial and pulmonary artery pressure in plain lignocaine group. They suggested that addition of adrenaline to lignocaine is associated with positive effects of adrenaline in preventing hypotension. But because of the potential hazards of increase in heart rate and cardiac output especially in elderly, the routine use of epidural adrenaline should be reconsidered in patients with high hemodynamic risk.

Silvestre et al[45] in 2011 carried out a prospective observational study to evaluate the safety of local anaesthesia during dental extraction in hypertensive patients. Blood pressure, heart rate and oxygen saturation were measured before and after administration of articaine with 4% epinephrine in one group and mepivacaine in another group. In the vasoconstrictor group, there was a rise in SBP and HR three minutes after the administration of LA. But it was not statistically significant. These values returned to normal after the procedure was completed. They concluded that during procedures such as dental extractions, no significant hemodynamic changes were seen in well controlled hypertensive patients attributable to anaesthetics with vasoconstrictors.

Ogunlewe et al[46] in 2011 conducted a prospective study to evaluate the changes in blood pressure and the pulse rate of patients with controlled hypertension receiving dental extraction under local anaesthesia using lignocaine with adrenaline, and to evaluate whether these changes were are attributable to addition of adrenaline. There was a increase in SBP following injection in both groups but fell to the same level that is slightly lower than the pre-anaesthetic value. The rise in BP was

significantly higher in plain lignocaine group which may be a result of impaired pain control from less effective anaesthesia with plain lignocaine.

Chaudhry et al[47] in 2011 evaluated the effect of epinephrine containing dental local anaesthetic on BP and pulse rate in hypertensive patients. All patients received 3.6 ml of 2% Lignocaine with 1:100,000 epinephrine. A decrease in systolic in stage 2 hypertension patients after 2 and 5 minutes of injections was noted. The DBP fell in all the groups after injections.

Sivanmalai et al[48] in 2012 conducted a prospective randomised clinical study to investigate the hemodynamic effects of adrenaline in lignocaine in clinical doses during third molar surgery under local anaesthesia. No significant change in heart rate or blood pressure was noted during the study. But there was a rise in plasma potassium and blood glucose concentration 20 min after administration of lignocaine with adrenaline.

Ketabi M et al[49] in 2012 conducted a randomized double-blind clinical trial comparing the changes in HR and BP after administration of lignocaine with and without epinephrine. In this study, the mean systolic and diastolic BP and HR was reduced after injection of lignocaine without epinephrine. After administration of lignocaine with adrenaline, the mean systolic and diastolic BP and HR was found to increase.

Daniel et al[50] in 2012 compare cardiovascular safety profiles of two dental anaesthetics, articaine versus standard mepivacaine solutions used during periodontal treatment in cardiovascular patients. A rise in HR, SBP and DBP were noticed in local anaesthetics with adrenaline group when compared to those without adrenaline. Oxygen saturation did not show any significant alterations. He concluded that the

administration of anaesthetics containing vasoconstrictors in patients with cardiovascular diseases should be done with caution.

Miratashi et al[51] in 2012 conducted a double blinded randomized clinical trial to evaluate hemodynamic effects of intraocular epinephrine irrigation during cataract surgery. Patients were randomly allocated into two groups with one group receiving intraocular irrigation fluid (balanced salt solution) with epinephrine 1:1,00,000 and the control group received plain intraocular irrigation fluid. Heart rate (HR), systolic and diastolic blood pressure (SBP, DBP) were measured before and at 5, 10, 15 minutes in both groups. DBP was decreased at 5 minutes after administration in the epinephrine group and increased at 10 and 15 minutes. There were no significant differences noticed between the two groups. So they concluded that the use of epinephrine 1: 1,00, 000 has minimal hemodynamic effects.

This is a prospective, randomised controlled study. Patients who reported to the Department of Oral and Maxillofacial Surgery, Sree Mookambika Institute of Dental Science, Kulasekharam K.K district, Tamilnadu were included in the study. Thirty patients who satisfied the required criteria formed the study sample.

INCLUSION CRITERIA:

- Patients willing to take part in the study.
- Patients who require multiple mandibular minor oral surgical procedures under local anaesthesia through inferior alveolar nerve block.
- Patients who fulfil ASA (American Society of Anaesthesiologists) Grade 1 criteria.
- Patients over 18 years of age.

EXCLUSION CRITERIA:

- Patients who do not fulfil ASA grade 1 criteria.
- Patients who have a positive history of pheochromocytoma, hyperthyroidism, asthma, hypertension or any other cardio respiratory disorders.
- Patients who are under tricyclic anti depressants, MAO inhibitors and anti hypertensive drugs.
- Patients with known allergy to local anaesthetic drugs.

MATERIALS USED

- Multi parameter monitor (BPL EXCELLO Eco, BPL -India)
- 3 ml single use syringe (Dispovan, Hindustan Syringes and Medical Devices, India)
- 26 gauge needle (Dispovan, Hindustan Syringes and Medical Devices, India)
- Lignocaine 2% (Zelcaine 2%, Bio Medica Laboratories, India)
- Lignocaine 2% adrenaline 1:80,000 (Lignox 2% A 1:80,000, Indoco Remedies, Indore, India)
- Lignocaine 2% adrenaline 1:2,00,000 (Xylocaine 2% Adrenaline (1:2,00,000) , Astra Zeneca, India)
- Topical lignocaine 2% (Lox 2% gel, Neon laboratories, India)

Zelcaine 2%,	**Lignoxox 2% adrenaline 1:80,000**	**Xylocaine 2% Adrenaline 1:2,00,000**
Lignocaine Hydrochloride 20.0 mg	Lignocaine Hydrochloride 24.64 mg	Lignocaine Hydrochloride 21.3 mg
Sodium Chloride 6mg	Adrenaline Bitartrate 0.0125 mg	Adrenaline Bitartrate 0.005 mg
Methylparaben 1.0 mg	Methylparaben 1 mg	Sodium metabisulphite 0.5mg
Water for injection 1.0ml	Water for injection	Sodium Chloride 6mg
		Water for injection

Procedure

All Patients who satisfied the pre requisites were well informed regarding conduct of the procedure, and an informed written consent was obtained. Using a repeated - measure design, each subject was administered with local anaesthesia prior to the desired procedure. The various anaesthetic solutions tested included plain Lignocaine 2%, Lignocaine 2% with adrenaline 1:80,000 and Lignocaine 2% with adrenaline 1: 2, 00,000 forming group I, .group II, group III respectively. The sequence of solution administration was determined randomly, and all the injections were performed by a single operator. During each appointment a single agent was administered and on subsequent appointments, the choice of agent differed in terms of their adrenaline concentrations. Every appointment was spaced one week apart, totalling 3 visits for each patient.

During each visit, prior to injection of local anaesthesia, systolic blood pressure, diastolic blood pressure, heart rate and oxygen saturation were noted using a multi-parameter monitor with the patient in a semi reclined position. The instrument was automated and had monitors for non invasive blood pressure and pulse oximetry. It had a measure strip of 10 mmHg to 300 mmHg for systolic blood pressure, 10 mmHg to 300mmHg for diastolic blood pressure, 10 beats/min to 300 beats/min for heart rate and 0% to 100% for oxygen saturation. The BP cuff was connected to one arm and pulse oximeter probe was connected to the other hand index finger.

All patients were categorised into groups based on the choice of local anaesthetic agent used with their respective concentration of adrenaline. The total no. of groups being three, group I included patients who received plain Lignocaine 2%,

group II received Lignocaine 2% with adrenaline 1:80,000 and group III received Lignocaine 2% with adrenaline 1:2, 00,000.

Before local anaesthetic administration, all patients received topical anaesthesia at the injection site using 2% lignocaine topical preparation. A standard inferior alveolar nerve block was administered using a disposable aspirating type syringe and 26 gauge long needle. Aspiration was performed after needle insertion and before deposition of anaesthetic solution to avoid inadvertent intravascular injection. Injection was done slowly at a rate of 1ml per minute. All patients received 1.8 ml of local anaesthetic solutions during each appointment.

Under each group, the following parameters such as oxygen saturation, heart rate systolic and diastolic blood pressure were recorded immediately after administration and at timely intervals of 5 min, 10 min, 15 min, 20 min and 30 min.

Case History Proforma

Name :

Age/Sex :

O.P.No :

Address :

Chief complaint :

History of Present Illness (HOPI) :

Past Medical History :

Past dental History :

History of drug allergy :

Habit :

Family history :

Extra/Intra oral Examination :

Provisional Diagnosis :

Treatment done :

Group I

SPO_2

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

HEART RATE

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

BLOOD PRESSURE

S.N	Before administration		Immediate		5 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1-30														

Group II

SPO_2

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

HEART RATE

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

BLOOD PRESSURE

S.N	Before administration		Immediate		5 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1-30														

Group III

SPO_2

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

HEART RATE

Sl.No.	Before Administration	Immediate	5min	10min	15min	20min	30min
1-30							

BLOOD PRESSURE

S.N	Before administration		Immediate		5 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1-30														

Figure I – Materials used

Figure II- Administration of local anaesthesia

The hemodynamic changes on administration of plain lignocaine and lignocaine with different concentrations of adrenaline were recorded using a multi parameter monitor. The SPO_2, SBP and DBP for Group-I, Group-II, Group-III at different time periods were recorded and tabulated as shown in table 1,2,3,4,5,6,7,8,9. The mean valued are shown in tables 10, 11, 12 and 13

Table-1: Effect of lignocaine 2% on SPO_2

Table-2: Effect of lignocaine 2% on Heart rate

Table-3: Effect of lignocaine 2% on Blood pressure

Table-4: Effect of lignocaine 2% with adrenaline 1:80,000 on SPO_2

Table-5: Effect of lignocaine 2% with adrenaline 1:80,000 on heart rate

Table-6: Effect of lignocaine dilution 1:80,000 blood pressure

Table-7: Effect of lignocaine 2% with adrenaline 1:2,00,000 on SPO_2 .

Table-8: Effect of lignocaine 2% with adrenaline 1:2,00,000 on heart rate

Table-9: Effect of lignocaine 2% with adrenaline 1:200000 on Blood pressure

Table-10: Mean values of SPO_2 in different groups

Table-11: Mean values of heart rate in different groups

Table-12: Mean values of systolic blood pressure in different groups

Table-13: Mean values of diastolic blood pressure

TABLES

Group-I : Lignocaine 2%

Table-1: Effect of Lignocaine 2% on SPO_2

S.NO	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	98	98	97	98	97	98	98
2	99	96	98	99	99	99	98
3	99	96	98	98	98	98	98
4	97	95	94	95	96	99	98
5	98	98	98	98	98	98	94
6	98	98	98	98	98	98	98
7	98	98	98	98	98	98	98
8	98	98	97	98	97	98	98
9	99	96	98	99	99	99	98
10	99	96	98	98	98	98	98
11	97	95	94	95	96	99	98
12	98	98	96	98	98	98	94
13	98	98	98	98	98	98	98
14	97	98	98	98	98	96	97
15	98	99	97	98	97	98	98
16	99	96	96	99	99	99	98
17	97	96	98	98	97	98	98
18	97	95	94	95	96	99	98
19	98	97	98	96	98	98	74
20	93	98	99	98	98	97	95
21	98	95	98	98	99	99	98
22	93	95	96	98	97	99	98
23	99	96	98	99	99	99	98
24	99	96	98	98	98	98	98
25	97	95	94	95	96	99	98
26	98	98	98	98	98	98	94
27	98	98	98	98	98	98	98
28	98	98	98	98	98	98	98
29	98	98	97	98	97	98	98
30	99	96	98	99	99	99	98

Table-2: Effect of Lignocaine 2% on Heart Rate

S.NO	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	58	50	58	59	59	61	66
2	99	91	89	86	88	98	98
3	74	71	86	73	71	74	73
4	77	75	74	75	76	79	78
5	83	81	84	79	84	83	80
6	98	98	102	102	103	105	101
7	83	81	84	84	79	83	80
8	58	50	59	59	59	61	66
9	98	91	89	86	88	98	98
10	75	71	85	73	71	74	73
11	78	75	74	74	76	79	78
12	82	81	84	79	84	83	80
13	99	98	102	102	103	105	100
14	75	79	85	84	80	81	80
15	58	50	58	59	59	61	65
16	99	91	90	87	88	98	99
17	75	71	86	74	71	75	74
18	77	75	74	75	76	79	78
19	82	81	85	78	84	81	80
20	98	98	100	102	103	105	101
21	81	81	82	85	79	83	79
22	54	49	58	59	59	67	65
23	98	91	89	87	82	98	99
24	77	71	86	73	71	73	74
25	78	75	74	75	77	79	78
26	80	80	84	78	84	83	79
27	99	99	102	102	103	105	101
28	74	75	85	85	79	80	81
29	59	49	58	59	60	63	66
30	90	90	89	86	88	99	99

Table-3: Effect of Lignocaine 2% on Blood pressure

.NO	Before Administration		Immediate		05 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1	120	70	111	64	114	74	117	73	114	70	112	66	111	65
2	118	83	123	90	120	86	120	87	122	86	117	78	116	83
3	117	72	115	72	122	80	116	70	117	74	114	72	115	74
4	103	69	104	64	105	65	102	68	103	58	102	65	101	65
5	123	81	131	80	117	77	124	76	118	73	114	80	125	82
6	127	79	121	77	133	84	116	79	126	77	120	79	117	76
7	125	81	133	80	119	77	126	76	120	73	124	83	124	82
8	120	70	111	64	114	74	117	73	115	70	112	66	111	65
9	119	84	123	90	120	86	120	87	122	86	117	78	116	83
10	117	72	114	73	122	80	116	70	117	74	114	72	115	74
11	103	69	104	64	104	65	102	68	103	58	102	65	102	65
12	123	81	131	80	117	77	125	76	119	74	115	80	125	82
13	127	79	121	77	133	84	116	79	126	77	120	78	117	76
14	125	74	121	72	130	78	126	76	126	70	121	72	120	70
15	120	70	111	64	114	74	117	73	114	70	112	66	111	65
16	118	83	123	90	120	86	120	87	122	86	117	78	116	82
17	117	72	115	72	122	80	116	70	117	74	114	72	115	73
18	104	69	105	64	105	65	102	68	103	58	102	65	101	60
19	122	81	131	80	117	77	124	76	118	73	114	80	125	82
20	127	79	123	77	133	84	116	79	126	77	120	79	117	75
21	124	81	133	80	119	77	126	76	120	74	124	83	125	82
22	120	70	112	64	114	74	117	73	115	70	112	66	111	65
23	121	70	111	64	114	74	117	73	114	69	113	66	112	64
24	119	83	123	90	120	86	120	87	122	86	117	78	116	83
25	117	72	114	72	120	80	117	70	118	74	115	70	114	74
26	103	69	104	64	105	65	102	68	103	58	102	65	100	64
27	123	81	131	80	117	77	124	76	118	73	114	80	125	89
28	127	79	121	77	133	84	116	79	126	77	120	79	118	76
29	125	80	133	80	119	77	126	76	120	73	124	83	124	82
30	120	70	111	64	114	74	117	73	115	70	112	66	112	64

Group-II: 1:80000

Table-4: Effect of lignocaine 2% with adrenaline 1:80000 on SPO_2

S.NO	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	98	98	98	98	98	99	98
2	98	96	97	97	98	96	98
3	98	98	96	98	97	98	98
4	98	98	99	98	98	98	98
5	98	93	98	98	97	98	98
6	99	98	99	98	99	99	98
7	98	98	98	98	98	98	98
8	98	98	98	98	98	98	98
9	98	98	98	98	98	98	98
10	98	98	98	98	98	98	98
11	99	99	99	98	99	99	98
12	99	96	99	99	95	99	96
13	98	98	98	98	98	98	98
14	98	98	97	98	98	98	98
15	98	98	98	98	98	98	98
16	98	98	97	98	98	98	98
17	99	98	99	99	99	99	98
18	99	98	99	99	99	100	99
19	98	97	97	98	98	98	98
20	97	98	96	96	96	94	96
21	97	98	98	94	98	98	96
22	98	97	97	98	98	99	99
23	98	98	97	98	99	98	96
24	98	98	98	98	98	98	98
25	99	98	98	99	99	99	98
26	99	98	99	99	99	99	99
27	98	98	97	98	98	98	98
28	97	98	97	97	96	94	96
29	98	98	98	97	98	98	96
30	98	99	99	99	98	98	98

Table-5: Effect of lignocaine 2% with adrenaline 1:80000 on heart rate

S.NO	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	57	60	67	58	61	69	58
2	91	87	88	84	82	89	84
3	69	58	64	76	73	75	74
4	82	83	92	92	85	79	87
5	81	84	80	82	96	78	82
6	102	107	88	98	94	98	93
7	79	81	82	83	84	79	80
8	55	56	69	89	54	53	54
9	86	75	70	88	85	84	83
10	85	87	90	91	96	89	89
11	92	97	88	98	94	98	98
12	90	99	99	99	98	100	98
13	96	78	70	98	85	94	93
14	76	75	85	83	78	78	75
15	85	87	90	90	96	89	89
16	76	75	85	83	78	78	78
17	80	89	89	89	90	98	97
18	75	74	80	89	74	75	84
19	51	55	54	51	56	55	52
20	98	83	86	91	91	85	88
21	95	95	88	88	70	90	84
22	91	89	84	80	79	87	86
23	69	62	69	73	70	71	74
24	82	82	83	90	84	79	82
25	79	89	92	84	89	78	81
26	99	100	93	92	94	98	90
27	80	84	80	79	83	79	82
28	64	72	67	88	89	53	55
29	68	79	72	83	82	84	82
30	59	83	89	92	96	89	84

Table-6: Effect of lignocaine dilution 1:80000 on blood pressure

S.No	Before Administration		Immediate		05 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1	131	84	122	81	127	80	119	71	121	81	118	73	110	72
2	113	87	110	66	113	73	111	70	109	70	111	66	103	63
3	114	74	107	69	122	71	112	67	116	62	117	69	118	79
4	108	74	109	71	112	75	108	72	103	63	106	56	107	61
5	125	89	122	87	119	78	122	84	111	78	121	80	115	74
6	123	81	120	75	122	75	134	84	125	74	135	77	125	68
7	121	74	118	84	119	76	113	74	109	63	113	73	115	67
8	122	82	120	69	112	65	119	66	122	69	118	69	121	73
9	120	75	121	78	121	80	120	67	122	71	119	69	123	69
10	123	70	110	67	112	70	122	61	128	71	120	73	119	72
11	124	81	121	75	122	78	130	75	125	74	131	77	128	68
12	134	82	134	80	137	83	137	86	126	81	117	80	116	77
13	120	75	122	78	133	80	117	67	120	71	118	69	123	69
14	122	71	117	69	116	65	130	84	122	68	120	73	121	62
15	123	70	108	66	111	70	122	61	123	71	120	73	119	69
16	123	71	118	69	116	65	130	84	123	68	121	73	122	62
17	135	82	132	80	139	83	138	86	126	81	117	80	116	77
18	119	70	118	76	108	61	108	62	104	62	110	67	100	67
19	151	71	154	77	163	81	178	76	169	77	154	78	145	72
20	133	77	132	71	130	73	141	74	135	63	134	59	130	66
21	130	80	120	80	125	82	118	70	121	81	118	73	110	72
22	110	83	119	65	111	73	111	70	109	70	111	66	103	63
23	100	77	110	68	121	71	112	68	118	65	117	69	118	79
24	102	72	110	70	110	75	108	72	103	63	105	56	107	61
25	120	85	120	86	118	77	122	84	111	78	121	79	115	74
26	122	82	122	74	120	76	134	84	125	74	135	77	124	68
27	128	70	119	82	110	75	113	74	109	63	113	73	115	67
28	120	80	121	70	115	64	119	66	122	69	118	69	121	73
29	122	76	120	77	128	82	120	67	122	71	119	69	123	70
30	199	72	119	65	110	69	122	61	128	71	120	73	119	72

Group-III: 1:200000

Table-7: Effect of Lignocaine 2% with adrenaline 1:200000 on SPO_2

Sl. No.	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	97	97	98	97	97	97	98
2	98	96	96	98	97	97	96
3	98	98	98	98	98	98	98
4	99	100	99	99	86	99	98
5	97	97	99	98	98	98	98
6	98	97	98	98	98	98	98
7	97	98	98	99	98	98	98
8	98	98	98	98	98	98	98
9	98	98	98	98	98	98	98
10	98	98	98	98	98	98	98
11	98	97	98	98	98	98	98
12	98	99	98	98	98	98	98
13	98	98	99	98	98	98	98
14	97	98	98	98	97	98	98
15	98	98	98	98	97	97	98
16	97	98	98	98	97	98	98
17	98	97	99	99	99	98	99
18	99	95	99	99	96	99	100
19	97	97	98	98	98	98	98
20	97	96	98	98	98	97	98
21	98	98	94	98	98	98	98
22	98	97	98	98	98	98	98
23	98	99	99	98	98	98	98
24	98	98	99	98	98	98	98
25	97	98	98	98	97	98	98
26	98	98	98	97	97	97	98
27	97	98	98	98	97	98	98
28	98	97	99	98	99	98	99
29	99	99	99	99	96	99	99
30	97	97	98	98	97	98	98

Table-8: Effect of lignocaine 2% with adrenaline 1:200000 on heart rate

S.NO	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
1	67	71	70	62	63	68	69
2	96	90	90	88	80	84	77
3	64	63	76	76	70	82	72
4	80	88	85	86	98	80	83
5	87	81	79	79	78	81	84
6	97	94	102	91	96	96	91
7	87	81	79	79	78	81	86
8	55	54	59	58	55	61	67
9	91	94	89	85	86	84	84
10	85	87	90	90	96	89	89
11	97	95	98	91	96	96	91
12	89	98	98	96	97	98	98
13	101	104	99	95	86	94	94
14	74	78	87	90	76	74	74
15	77	75	80	78	77	72	80
16	74	78	84	89	78	74	74
17	81	86	84	80	81	85	84
18	79	78	72	74	81	79	75
19	50	55	55	51	50	51	51
20	88	83	80	82	78	81	86
21	67	71	72	62	63	68	69
22	96	90	89	88	80	84	77
23	64	63	76	76	74	82	72
24	80	88	85	86	98	82	83
25	87	81	79	79	78	81	84
26	97	94	99	91	96	96	91
27	87	81	79	79	78	81	86
28	55	55	59	58	55	61	67
29	91	93	89	84	86	84	84
30	85	84	90	99	96	89	89

Table-9: Effect of lignocaine 2% with adrenaline 1:200000 on Blood Pressure

S.No	Before Administration		Immediate		05 min		10 min		15 min		20 min		30 min	
	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1	119	75	113	67	118	75	118	74	116	67	115	72	115	68
2	132	89	128	77	121	78	121	85	115	87	113	74	107	74
3	114	69	103	61	112	66	129	72	113	65	121	71	124	68
4	109	74	112	83	110	68	117	72	114	73	114	69	109	68
5	137	86	124	82	118	75	113	71	123	99	117	69	116	84
6	126	77	126	85	131	66	128	81	121	77	122	76	117	73
7	139	86	126	82	120	75	115	71	123	79	119	69	118	84
8	110	57	101	55	109	64	107	58	108	64	107	57	112	64
9	130	88	144	74	129	85	134	85	124	80	122	87	119	72
10	116	71	114	71	117	72	118	64	112	63	110	75	109	64
11	127	77	124	85	131	66	129	81	122	77	128	76	117	73
12	121	75	115	67	118	75	120	74	116	75	115	72	115	68
13	130	88	124	73	131	85	137	85	124	82	122	87	119	74
14	124	77	124	85	131	66	120	81	121	76	122	77	117	73
15	116	71	115	71	117	72	115	64	112	63	108	65	110	75
16	124	77	121	85	131	66	131	84	121	76	122	77	119	73
17	122	75	116	74	119	75	121	74	117	75	116	72	116	74
18	110	68	115	66	125	73	111	68	105	75	114	75	108	72
19	150	73	159	80	168	91	153	73	139	66	143	76	140	71
20	137	86	124	82	118	75	133	75	135	75	135	74	134	78
21	119	77	113	67	118	75	118	74	116	67	115	72	114	66
22	132	82	128	77	121	78	122	86	115	87	113	74	107	74
23	115	69	103	61	112	66	129	72	113	65	121	71	124	68
24	109	74	112	83	110	68	117	74	114	73	114	69	109	68
25	136	86	124	82	118	75	115	71	123	99	117	69	116	84
26	125	77	126	85	131	66	128	81	121	77	122	76	117	73
27	139	86	126	82	120	75	115	72	123	79	118	70	116	84
28	116	57	106	57	109	64	107	58	108	64	107	57	112	64
29	130	88	144	74	119	85	124	85	126	79	124	87	119	72
30	116	71	114	71	117	72	118	64	112	63	110	75	109	64

Table-10: Mean values of SPO_2 in different groups

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	95.73 ±6.51	94.80 ±6.81	95.17 ±7.24	95.70 ±7.06	95.73 ±6.73	96.27 ±5.89	94.67 ±7.19
Group-II (1:80000)	98.13 ±0.57	97.70 ±1.09	97.87 ±0.90	97.93 ±0.94	98.00 ±1.29	97.70 ±0.91	97.70 ±0.92
Group-III (1:200000)	97.77 ±0.63	97.63 ±0.99	98.10 ±0.99	98.10 ±0.48	97.23 ±2.24	97.93 ±0.52	98.10 ±0.61

Table-11: Mean values of heart rate in different groups

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	80.53 ±13.91	77.27 ±15.28	81.83 ±13.09	79.30 ±12.76	79.47 ±13.10	83.10 ±13.90	82.30 ±12.49
Group-II (1:80000)	79.73 ±13.60	80.83 ±13.16	81.10 ±10.65	85.37 ±10.57	82.87 ±11.90	81.70 ±12.72	81.20 ±12.32
Group-III (1:200000)	80.93 ±13.78	81.10 ±13.12	82.43 ±11.92	80.73 ±12.10	80.13 ±13.44	80.60 ±11.04	80.37 ±10.02

Table-12: Mean values of systolic blood pressure

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	119.13 ±7.12	118.80 ±9.43	118.53 ±8.11	117.33 ±7.13	117.30 ±6.85	114.53 ±6.21	115.23 ±7.38
Group-II (1:80000)	124.57 ±17.22	119.83 ±9.31	120.73 ±11.47	123.00 ±14.01	120.23 ±12.37	119.90 ±9.76	117.70 ±9.03
Group-III (1:200000)	124.33 ±10.50	120.80 ±12.52	121.63 ±11.27	122.10 ±9.70	118.40 ±7.81	118.20 ±7.80	116.13 ±7.35

Table-13: Mean values of diastolic blood pressure

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	75.77 ±5.54	74.30 ±8.96	77.37 ±6.39	75.40 ±5.76	72.73 ±7.69	73.67 ±6.64	74.07 ±8.15
Group-II (1:80000)	77.23 ±5.67	74.17 ±6.47	74.20 ±6.13	72.90 ±8.16	70.77 ±6.17	71.27 ±6.39	77.70 ±9.03
Group-III (1:200000)	76.87 ±8.51	74.80 ±8.10	73.07 ±6.95	74.30 ±7.91	74.90 ±9.59	73.00 ±6.82	72.23 ±5.96

STATISTICAL ANALYSIS OF THE RESULTS

The statistical analysis was performed using SPSS software(statistical package for social sciences)version16.0. The data was interpreted at a confidence interval of 95%. One way ANOVA applied for statistical significance between the groups. Post hoc test followed by sheff test was used for multiple comparison to find statistical significance. P value < 0.05 was considered statistically significant.

Table-14: Comparison of SPO_2 values before administration with different time intervals within each groups

No significant changes are seen within the groups at different time periods.

Table -15: Multiple comparison of SPO_2 values with different time intervals within each groups

No significant changes are seen within the groups at different time periods.

Table-16: Comparison of heart rate before administration and at different time intervals within each group.

Statistical analysis in Group 1 shows minor variations in HR which was not significant.. Group II shows a significant increase in heart rate at 10 min. by 5 beats per min.

Table -17: Multiple comparison of heart rate with different time intervals within each groups.

Group 1 shows minor variations in HR which was not significant.. Group II shows a significant increase in heart rate at 10 min. by 5 beats per min.

Table-18: Comparison of systolic blood pressure before administration with different time intervals within each groups.

Group II shows a significant decrease in SBP immediately after administration by 5 mm Hg, at 20 min period by 5 mm Hg and at 30 min period by 7 mm Hg. Group III shows significant decrease in heart rate at 15 min and 20 min by 6 mm Hg and at 30 min by 7 mm Hg.

Table -19: Multiple comparison of systolic blood pressure with different time intervals within each group.

Group II shows a significant decrease in SBP immediately after administration by 5 mm Hg, at 20 min period by 5 mm Hg and at 30 min period by 7 mm Hg. Group III shows significant decrease in heart rate at 15 min and 20 min by 6 mm Hg and at 30 min by 7 mm Hg.

Table-20: Comparison of diastolic blood pressure before administration with different time intervals within each group.

Group II shows significant decrease in DBP by 6 mmHg at 15 min period and by 6 mmHg at 20 min period. Group III shows significant decrease in DBP by 5 mmHg at 30 min period.

Table -21: Multiple comparison of diastolic blood pressure with different time intervals within each group.

Group II shows significant decrease in DBP by 6 mmHg at 15 min period and by 6 mmHg at 20 min period. Group III shows significant decrease in DBP by 5 mmHg at 30 min period.

Table-22: Comparison of mean values of SPO_2 of Group-I with other groups at different

No significant changes are seen between the groups at different time periods.

Table-23: Multiple comparison of mean values of SPO_2 of groups at different time period

No significant changes are seen between the groups at different time periods.

Table-24 Comparison of mean values of heart rate of Group-I with other groups at different time periods

Shows significant increase in HR in Group II by 5 beats /min immediately after administration.

Table-25: Multiple comparison of mean values of heart rate of groups at different time periods

Shows significant increase in HR in Group II by 5 beats / min immediately after administration.

Table-26: Comparison of mean values of systolic blood pressure of Group-I with other groups at different time periods

Shows significant increase in SBP by 6 mmHg in group II at 10 min period and 5 mm Hg at 20 min . There is a significant increase in SBP by 3 mmHg in group III at 5 min period.

Table-27: Multiple comparisons of mean values of systolic blood pressure of groups at different time periods.

Shows significant increase in SBP by 6 mmHg in group II at 10 min period and 5 mm Hg at 20 min . There is a significant increase in SBP by 3 mmHg in group III at 5 min period

Table-28: Comparison of mean values of diastolic blood pressure of Group-I with other groups at different time periods.

No significant changes are seen between the groups at different time periods.

Table-29: Multiple comparison of mean values of diastolic blood pressure of groups at different time periods

No significant changes are seen between the groups at different time periods.

Graph-1: Comparison of SPO_2 values in Group-I (Lignocaine)

Shows no significant changes in SPO_2

Graph-2: Comparison of SPO_2 values in Group-II (1:80000 dilution)

Shows no significant changes in SPO_2

Graph-3: Comparison of SPO_2 values in Group-III (1:200000 dilution)

Shows no significant changes in SPO_2

Graph-4: Multiple comparison of SPO_2 values with different time intervals within the groups

Shows no significant changes in SPO_2

Graph-5: Comparison of heart rate in Group-I (Lignocaine) .

Group 1 shows no significant changes ih HR.

Graph-6: Comparison of heart rate in Group-II (1:80000 dilutions) .

Group II shows a significant increase in heart rate at 10 min

Graph-7: Comparison of heart rate in Group-III (1:200000 dilutions).

Shows no significant changes in HR

Graph-8: Multiple comparison of heart rate with different time intervals within the groups

Group 1 shows significant decrease in heart rate at 10 min. Group II shows a significant increase in heart rate at 10 min.

Graph-9: Comparison of systolic blood pressure in Group-I (Lignocaine 2%)

Graph-10: Comparison of systolic blood pressure in Group-II (1:80000 dilutions)

Group II shows a significant decrease in SBP immediately after administration, at 20min and 30min

Graph-11: Comparison of systolic blood pressure in Group-III (1:200000 dilutions)

Group III shows significant decrease in heart rate at 15 min, 20 min and at 30 min.

Graph-12: Multiple comparison of systolic blood pressure with different time intervals within the groups

Group II shows a significant decrease in SBP immediately after administration, at 20min and 30min. Group III shows significant decrease in heart rate at 15 min, 20 min and at 30 min.

Graph-13: Comparison of diastolic blood pressure in Group-I (Lignocaine 2%)

Graph-14: Comparison of diastolic blood pressure in Group-II (1:80000 dilution)

Group II shows significant decrease in DBP at 15 min period and at 20 min period.

Graph-15: Comparison of diastolic blood pressure in Group-III (1:200000 dilutions)

Group III shows significant decrease in DBP by at 30 min period.

Graph-16: Multiple comparison of diastolic blood pressure with different time intervals within each groups.

Group II shows significant decrease in DBP at 15 min period and at 20 min period. Group III shows significant decrease in DBP by at 30 min period.

TABLES

Table-14: Comparison of SPO_2 values before administration with different time intervals within each group

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	95.73±6.51	98.13±0.57	97.77±0.63
Immediate	94.80±6.81	97.70±1.09	97.63±0.99
5 min	95.17±7.24	97.87±0.90	98.10±0.99
10 min	95.70±7.06	97.93±0.94	98.10±0.48
15 min	95.73±6.73	98.00±1.29	97.23±2.24
20 min	96.27±5.89	97.70±0.91	97.93±0.52
30 min	94.67±7.19	97.70±0.92	98.10±0.61

Table -15: Multiple comparison of SPO_2 values with different time intervals within each group

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	95.73±6.51	98.13±0.57	97.77±0.63
Immediate	94.80±6.81	97.70±1.09	97.63±0.99
5 min	95.17±7.24	97.87±0.90	98.10±0.99
10 min	95.70±7.06	97.93±0.94	98.10±0.48
15 min	95.73±6.73	98.00±1.29	97.23±2.24
20 min	96.27±5.89	97.70±0.91	97.93±0.52
30 min	94.67±7.19	97.70±0.92	98.10±0.61

Table-16: Comparison of heart rate before administration and at different time intervals within each group.

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	80.53±13.91	79.73±13.60	80.93±13.78
Immediate	77.27±15.28	80.83±13.16	81.10±13.12
5 min	81.83±13.09	81.10±10.65	82.43±11.92
10 min	79.30±12.76	85.37±10.57*	80.73±12.10
15 min	79.47±13.10	82.87±11.90	80.13±13.44
20 min	83.10±13.90	81.70±12.72	80.60±11.04
30 min	82.30±12.49	81.20±12.32	80.37±10.02

(*P<0.05 significant compared heart rate before administration with 10 min)

Table -17: Multiple comparison of heart rate with different time intervals within each group.

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before administration	80.53±13.19	79.73±13.60	80.93±13.78
Immediate	77.27±15.28	80.83±13.16	81.10±13.12
5 min	81.83±13.09	81.10±10.65	82.43±11.92
10 min	70.39±12.76*,#,†	85.37±10.57*	80.73±12.10
15 min	79.47±13.10$	82.87±11.90	80.13±13.44
20 min	83.10±13.90$	81.70±12.72	80.60±11.04
30 min	82.30±12.49$	81.20±12.32	80.37±10.02

(*P<0.05 significant compared hear rate before administration with other groups, #P<0.05 significant compared heart rate immediate with other groups, †P<0.05 significant compared heart rate 5 min with other groups, $P<0.05 significant compared heart rate 10 min with other groups)

Table-18: Comparison of systolic blood pressure before administration with different time intervals within each groups

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	119.13±7.12	124.57±17.22	124.33±10.50
Immediate	118.80±9.43	119.83±9.31*	120.80±12.52
5 min	118.53±8.11	120.73±11.47	121.63±11.27
10 min	117.33±7.13	123.00±14.01	122.10±9.70
15 min	117.30±6.85	120.23±12.37	118.40±7.81*
20 min	114.53±6.21	119.90±9.76*	118.20±7.80*
30 min	115.23±7.38	117.70±9.03*	116.13±7.35*

(*P<0.05 significant compared systolic blood pressure before administration with other groups)

Table -19: Multiple comparison of systolic blood pressure with different time intervals within each groups

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	119.13±7.12	124.57±17.22	124.33±10.50
Immediate	118.80±9.43	119.83±9.31*	120.80±12.52
5 min	118.53±8.11	120.73±11.47	121.63±11.27
10 min	117.33±7.13	123.00±14.01	122.10±9.70
15 min	117.30±6.85	120.23±12.37	118.40±7.81*
20 min	114.53±6.21	119.90±9.76*,#	118.20±7.80*
30 min	115.23±7.38	117.70±9.03*,#	116.13±7.35*

(*P<0.05 significant compared systolic blood pressure before administration with other groups, #P<0.05 significant compared systolic blood pressure 10 min with other groups)

Table-20: Comparison of diastolic blood pressure before administration with different time intervals within each groups

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	75.77±5.54	77.23±5.67	76.87±8.51
Immediate	74.30±8.96	74.17±6.47	74.80±8.10
5 min	77.37±6.39	74.20±6.13	73.07±6.95
10 min	75.40±5.76	72.90±8.16	74.30±7.91
15 min	72.73±7.69	70.77±6.17*	74.90±9.59
20 min	73.67±6.64	71.27±6.39*	73.00±6.82
30 min	74.07±8.15	77.70±9.03	72.23±5.96*

(*P<0.05 significant compared diastolic blood pressure before administration with other groups)

Table -21: Multiple comparison of diastolic blood pressure with different time intervals within each groups

Groups	Group-I (MEAN±SD)	Group-II (MEAN±SD)	Group-III (MEAN±SD)
Before Administration	75.77±5.54	77.23±5.67	76.87±8.51
Immediate	74.30±8.96	74.17±6.47	74.80±8.10
5 min	77.37±6.39	74.20±6.13	73.07±6.95
10 min	75.40±5.76	72.90±8.16	74.30±7.91
15 min	72.73±7.69	70.77±6.17*	74.90±9.59
20 min	73.67±6.64	71.27±6.39*	73.00±6.82
30 min	74.07±8.15	77.70±9.03	72.23±5.96*

(*P<0.05 significant compared diastolic blood pressure before administration with other groups)

Table-22: Comparison of mean values of SPO_2 of Group-I with other groups at different time period

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	95.73 ±6.51	94.80 ±6.81	95.17 ±7.24	95.70 ±7.06	95.73 ±6.73	96.27 ±5.89	94.67 ±7.19
Group-II (1:80000)	98.13 ±0.57	97.70 ±1.09	97.87 ±0.90	97.93 ±0.94	98.00 ±1.29	97.70 ±0.91	97.70 ±0.92
Group-III (1:200000)	97.77 ±0.63	97.63 ±0.99	98.10 ±0.99	98.10 ±0.48	97.23 ±2.24	97.93 ±0.52	98.10 ±0.61

Table-23: Multiple comparison of mean values of SPO_2 of groups at different time period

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	95.73 ±6.51	94.80 ±6.81	95.17 ±7.24	95.70 ±7.06	95.73 ±6.73	96.27 ±5.89	94.67 ±7.19
Group-II (1:80000)	98.13 ±0.57	97.70 ±1.09	97.87 ±0.90	97.93 ±0.94	98.00 ±1.29	97.70 ±0.91	97.70 ±0.92
Group-III (1:200000)	97.77 ±0.63	97.63 ±0.99	98.10 ±0.99	98.10 ±0.48	97.23 ±2.24	97.93 ±0.52	98.10 ±0.61

Table-24 Comparison of mean values of heart rate of Group-I with other groups at different time periods

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	80.53 ±13.91	77.27 ±15.28	81.83 ±13.09	79.30 ±12.76	79.47 ±13.10	83.10 ±13.90	82.30 ±12.49
Group-II (1:80000)	79.73 ±13.60	81.83 ±13.16*	81.10 ±10.65	85.37 ±10.57	82.87 ±11.90	81.70 ±12.72	81.20 ±12.32
Group-III (1:200000)	80.93 ±13.78	80.10 ±13.12	82.43 ±11.92	80.73 ±12.10	80.13 ±13.44	80.60 ±11.04	80.37 ±10.02

(*P<0.05 significant compared heart rate at immediate between group-I and group-II)

Table-25: Multiple comparison of mean values of heart rate of groups at different time periods

Groups	Before administration	Immediate	5 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	80.53 ±13.91	77.27 ±15.28	81.83 ±13.09	79.30 ±12.76	79.47 ±13.10	83.10 ±13.90	82.30 ±12.49
Group-II (1:80000)	79.73 ±13.60	81.83 ±13.16*	81.10 ±10.65	85.37 ±10.57	82.87 ±11.90	81.70 ±12.72	81.20 ±12.32
Group-III (1:200000)	80.93 ±13.78	80.10 ±13.12	82.43 ±11.92	80.73 ±12.10	80.13 ±13.44	80.60 ±11.04	80.37 ±10.02

(*P<0.05 significant compared heart rate at immediate between group-I and group-II)

Table-26: Comparison of mean values of systolic blood pressure of Group-I with other groups at different time periods

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	119.13 ±7.12	118.80 ±9.43	118.53 ±8.11	117.33 ±7.13	117.30 ±6.85	114.53 ±6.21	115.23 ±7.38
Group-II (1:80000)	124.57 ±17.22	119.83 ±9.31	120.73 ±11.47	123.00 ±14.01*	120.23 ±12.37	119.90 ±9.76*	117.70 ±9.03
Group-III (1:200000)	124.33 ±10.50	120.80 ±12.52	121.63 ±11.27*	122.10 ±9.70*	118.40 ±7.81	118.20 ±7.80	116.13 ±7.35

(*P<0.05 significant compared systolic blood pressure group-I with other groups)

Table-27: Multiple comparison of mean values of systolic blood pressure of groups at different time periods

Groups	Before Administration	Immediate	05 min	10 min	15 min	20min	30 min
Group-I (Lignocaine)	119.13 ±7.12	118.80 ±9.43	118.53 ±8.11	117.33 ±7.13	117.30 ±6.85	114.53 ±6.21	115.23 ±7.38
Group-II (1:80000)	124.57 ±17.22	119.83 ±9.31	120.73 ±11.47	123.00 ±14.01*	120.23 ±12.37	119.90 ±9.76*	117.70 ±9.03
Group-III (1:200000)	124.33 ±10.50	120.80 ±12.52	121.63 ±11.27*	122.10 ±9.70*	118.40 ±7.81	118.20 ±7.80	116.13 ±7.35

(*P<0.05 significant compared systolic blood pressure group-I with other groups)

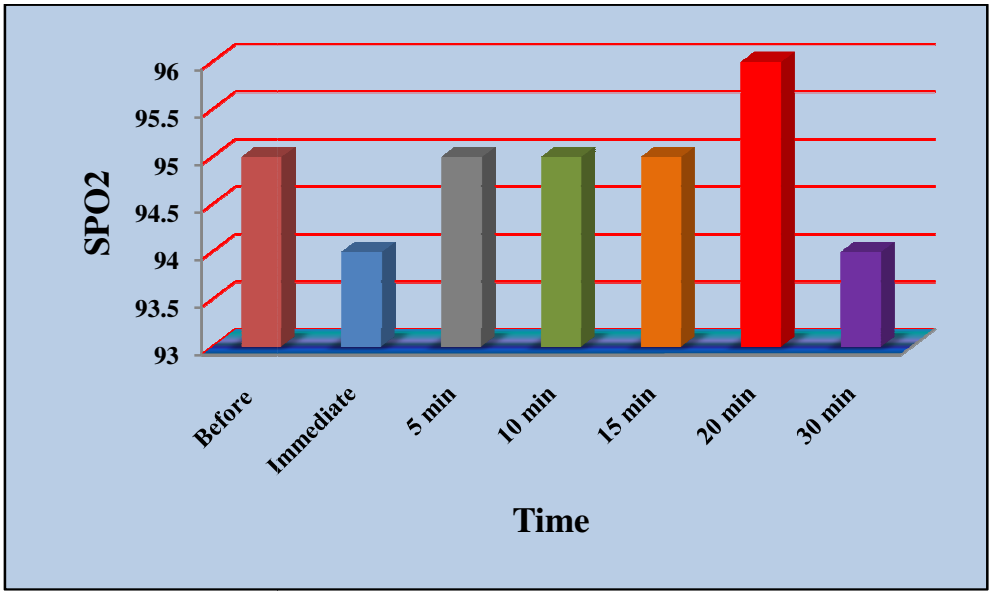
Table-28: Comparison of mean values of diastolic blood pressure of Group-I with other groups at different time periods

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	75.77 ±5.54	74.30 ±8.96	77.37 ±6.39	75.40 ±5.76	72.73 ±7.69	73.67 ±6.64	74.07 ±8.15
Group-II (1:80000)	77.23 ±5.67	74.17 ±6.47	74.20 ±6.13	72.90 ±8.16	70.77 ±6.17	71.27 ±6.39	77.70 ±9.03
Group-III (1:200000)	76.87 ±8.51	74.80 ±8.10	73.07 ±6.95	74.30 ±7.91	74.90 ±9.59	73.00 ±6.82	72.23 ±5.96

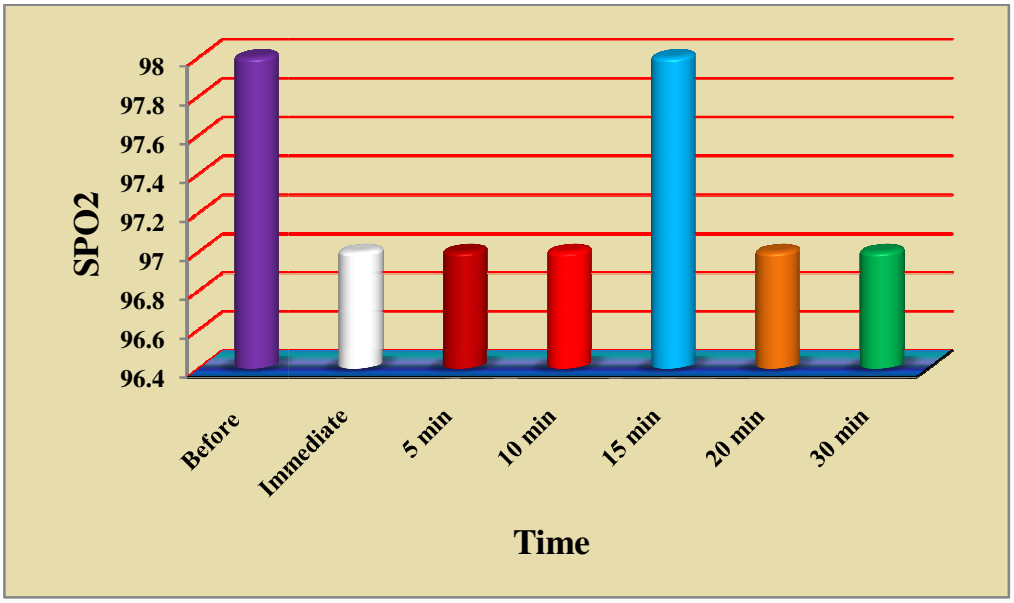
Table-29: Multiple comparison of mean values of diastolic blood pressure of groups at different time periods

Groups	Before Administration	Immediate	05 min	10 min	15 min	20 min	30 min
Group-I (Lignocaine)	75.77 ±5.54	74.30 ±8.96	77.37 ±6.39	75.40 ±5.76	72.73 ±7.69	73.67 ±6.64	74.07 ±8.15
Group-II (1:80000)	77.23 ±5.67	74.17 ±6.47	74.20 ±6.13	72.90 ±8.16	70.77 ±6.17	71.27 ±6.39	77.70 ±9.03
Group-III (1:200000)	76.87 ±8.51	74.80 ±8.10	73.07 ±6.95	74.30 ±7.91	74.90 ±9.59	73.00 ±6.82	72.23 ±5.96

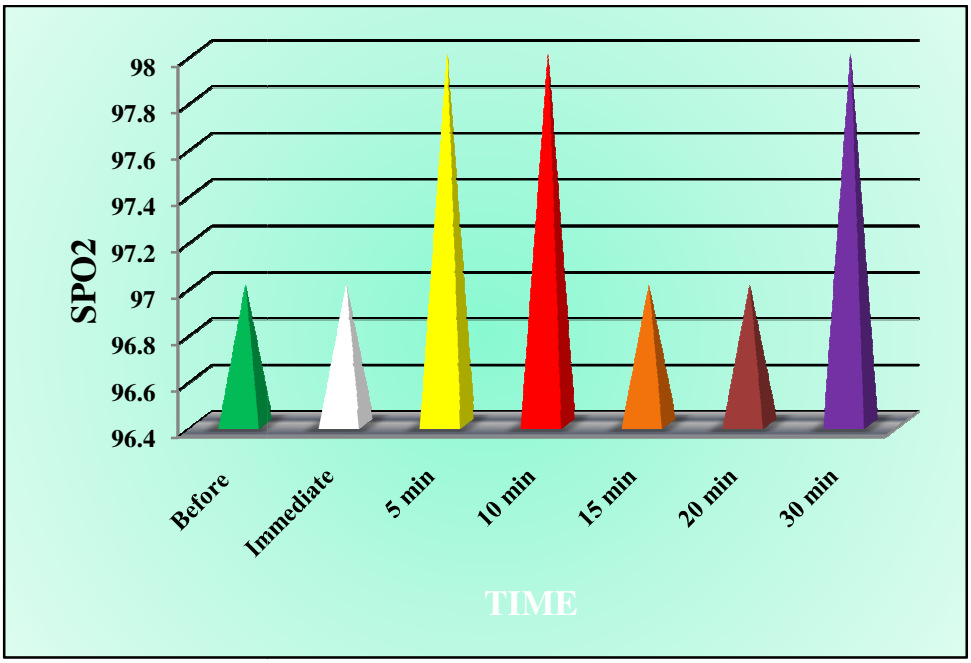
Graph-1: Comparison of SPO_2 values in Group-I (Lignocaine)



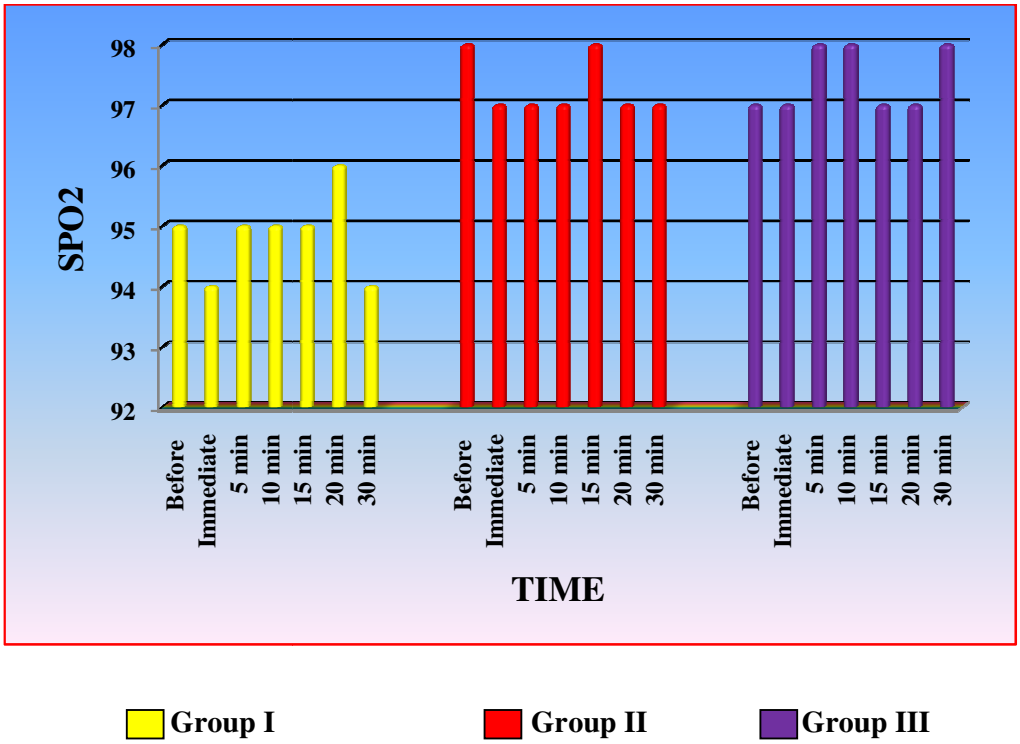
Graph-2: Comparison of SPO_2 values in Group-II (1:80000 dilution)



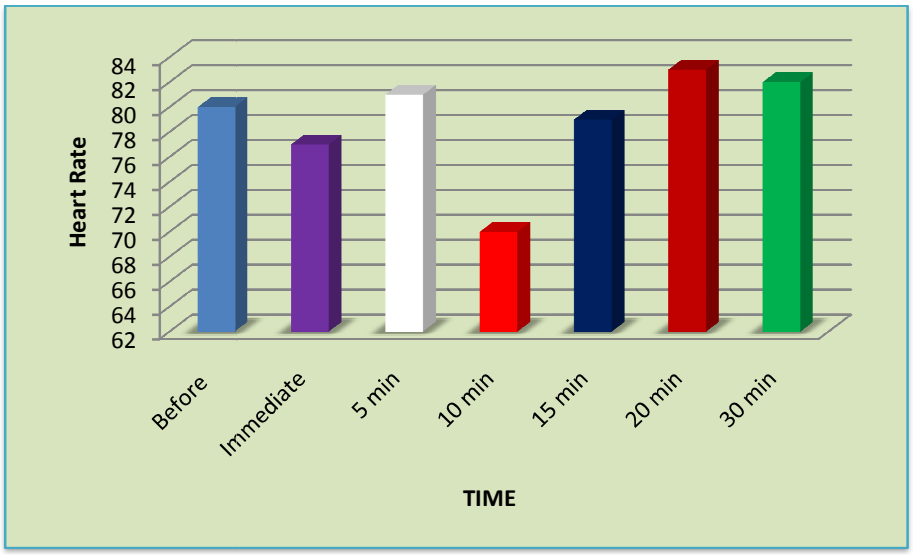
Graph-3: Comparison of SPO_2 values in Group-III (1:200000 dilution)



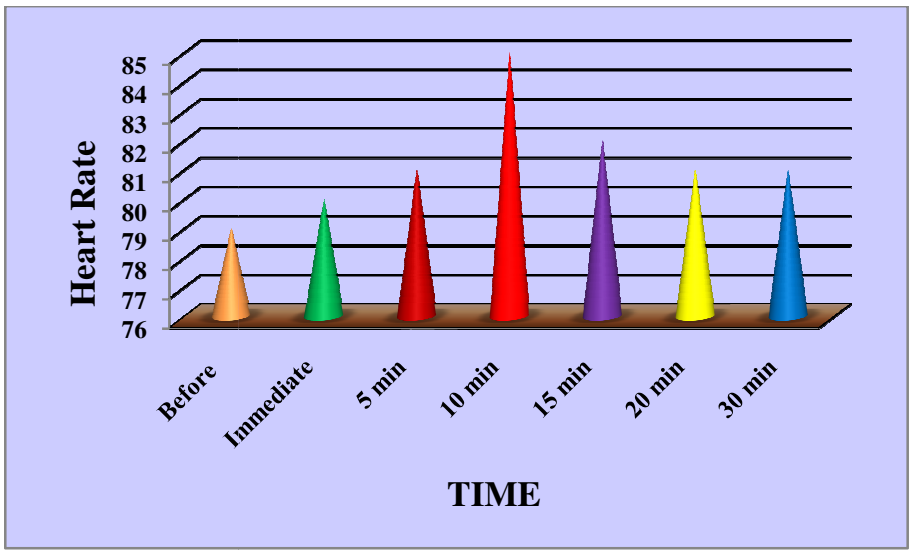
Graph-4: Multiple comparison of SPO_2 values with different time intervals within the groups



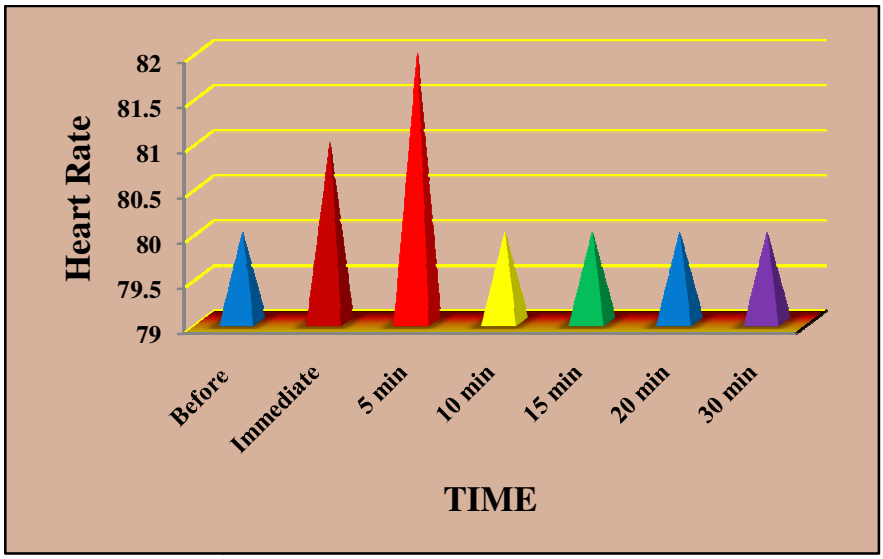
Graph-5: Comparison of heart rate in Group-I (Lignocaine)



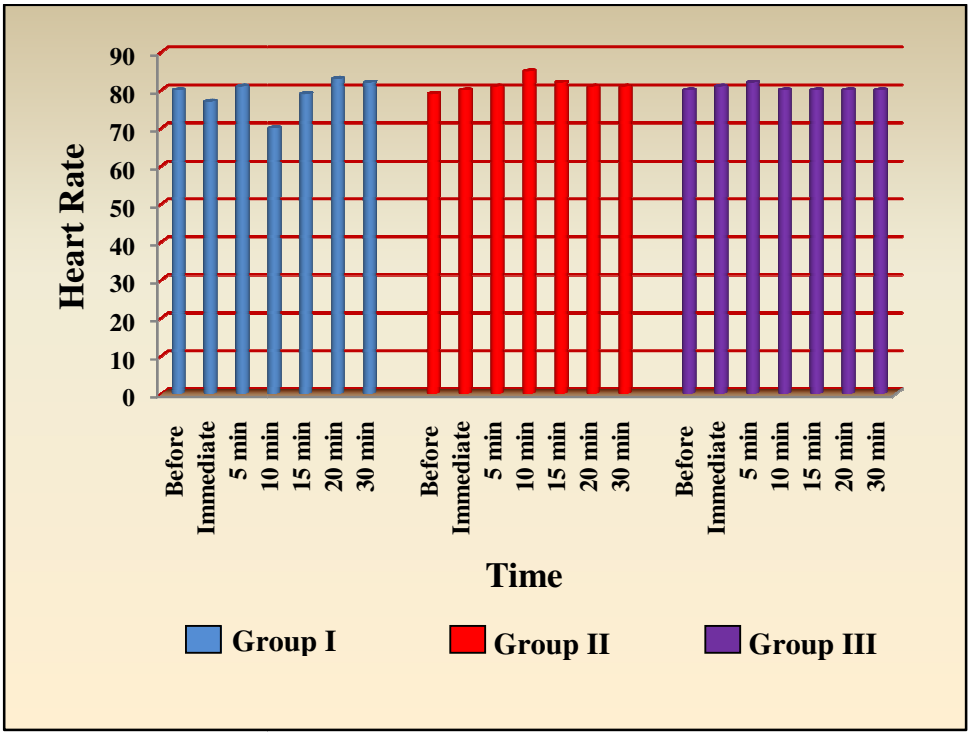
Graph-6: Comparison of heart rate in Group-II (1:80000 dilution)



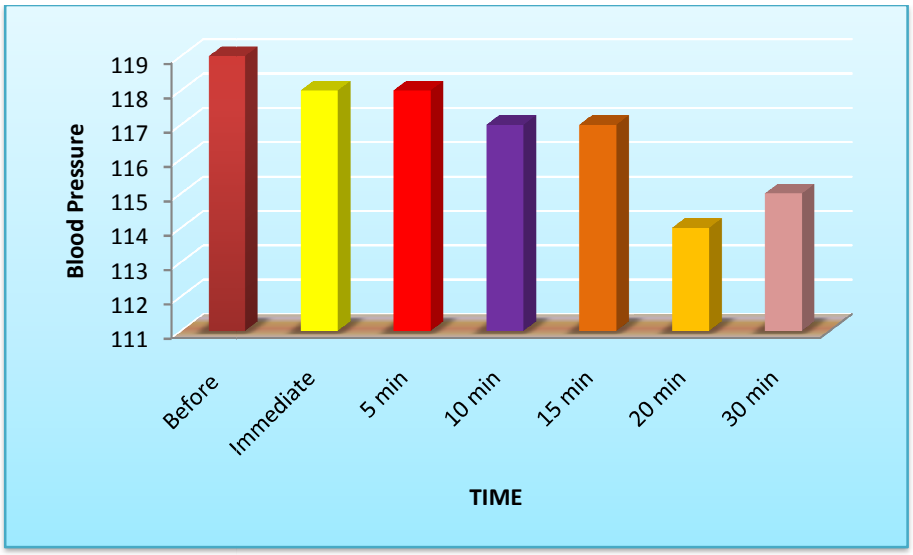
Graph-7: Comparison of heart rate in Group-III (1:200000 dilution)



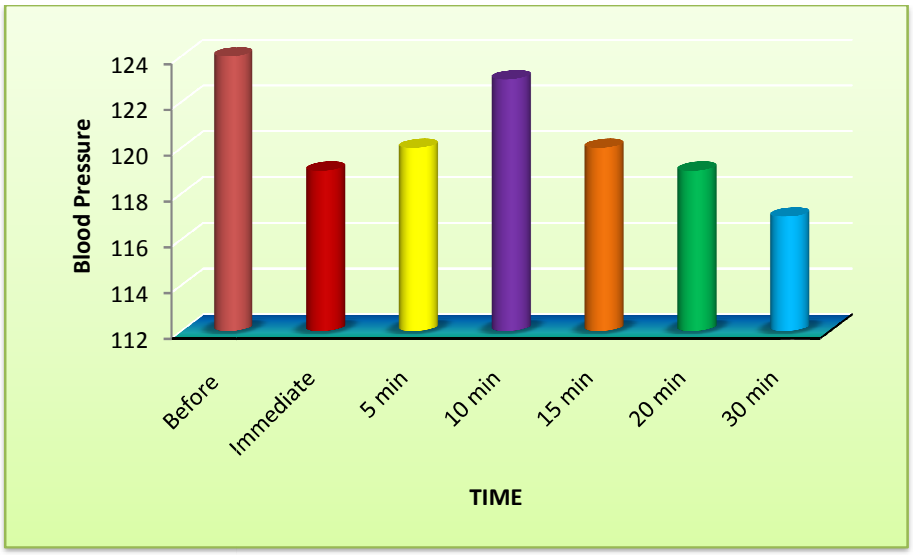
Graph-8: Multiple comparison of heart rate with different time intervals within the groups



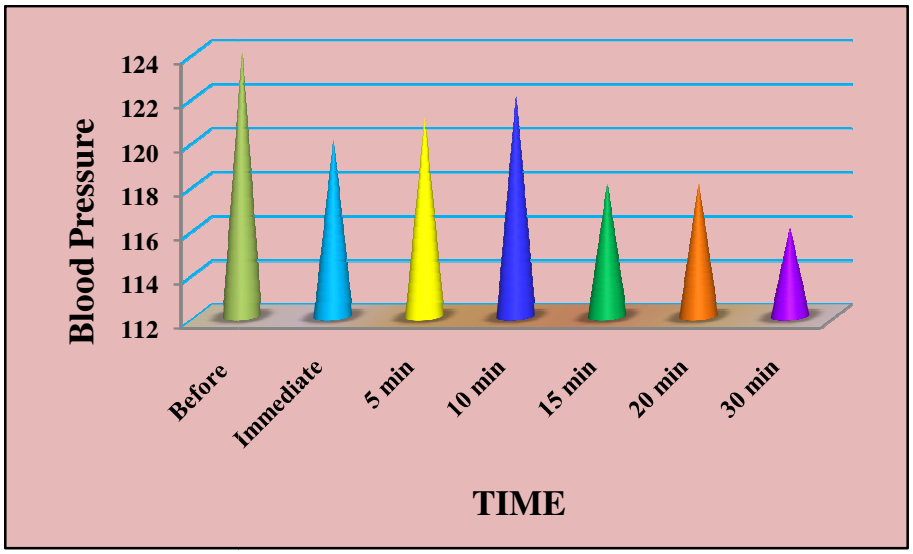
Graph-9: Comparison of systolic blood pressure in Group-I (Lignocaine)



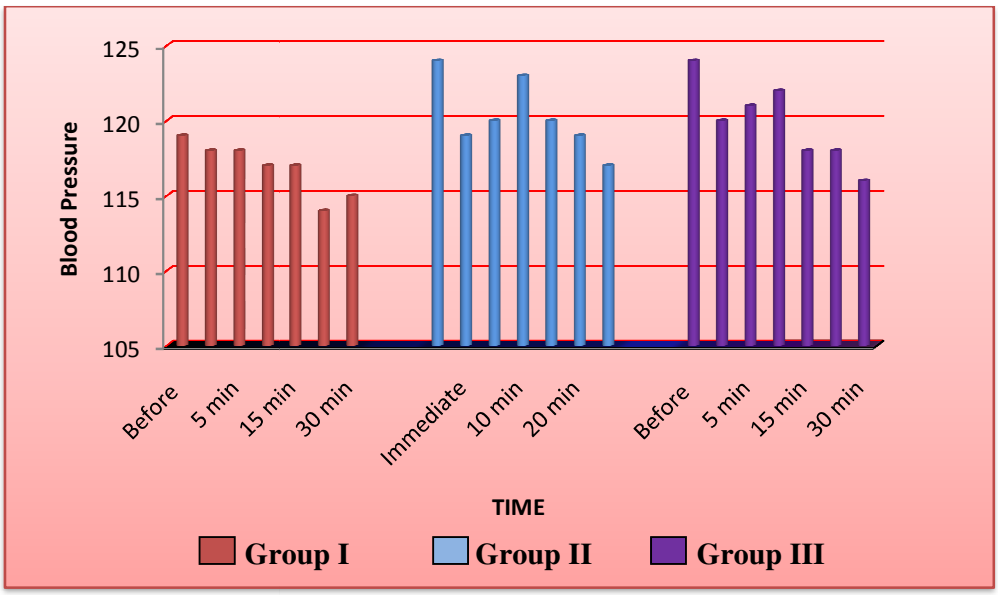
Graph-10: Comparison of systolic blood pressure in Group-II (1:80000 dilution)



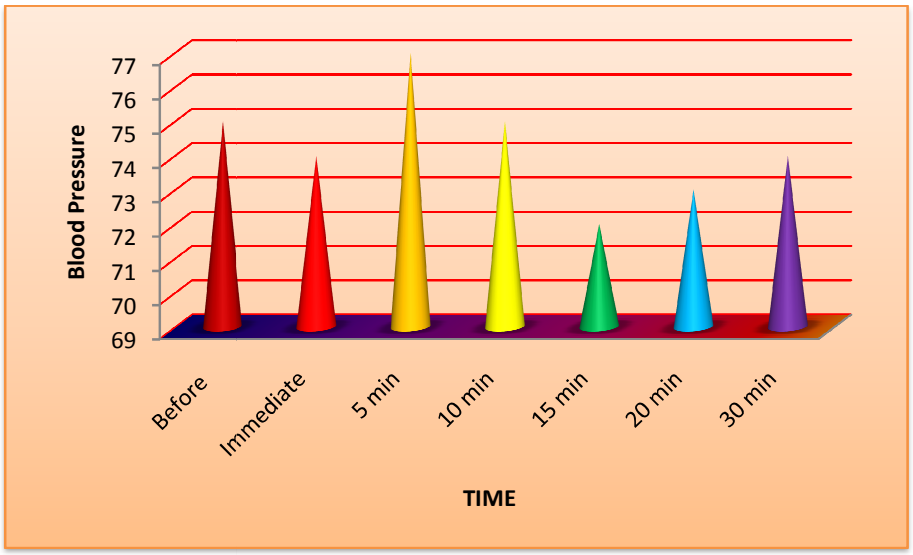
Graph-11: Comparison of systolic blood pressure in Group-III (1:200000 dilution)



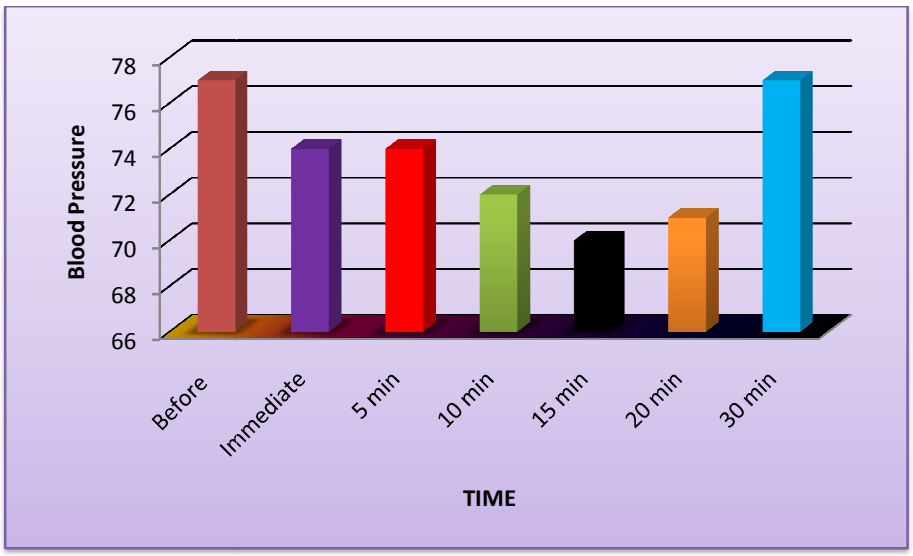
Graph-12: Multiple comparison of systolic blood pressure with different time intervals within the groups



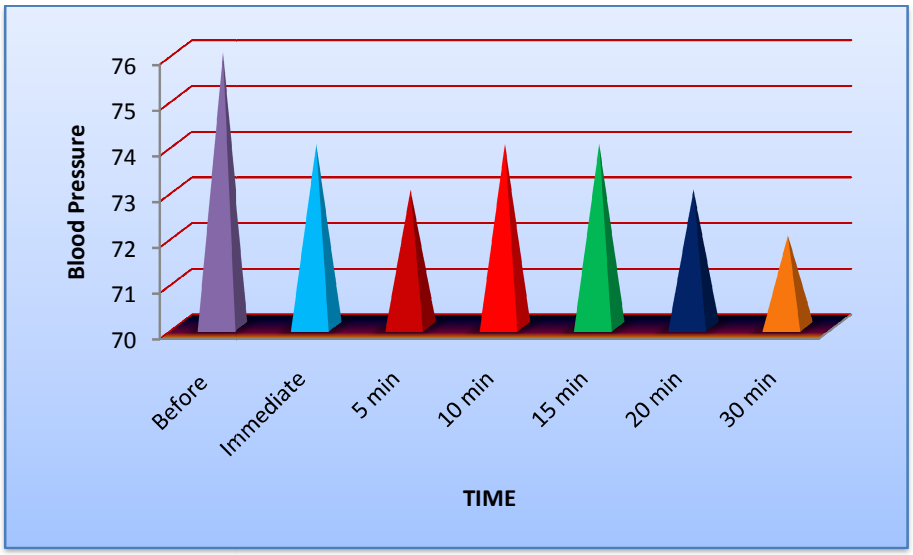
Graph-13: Comparison of diastolic blood pressure in Group-I (Lignocaine)



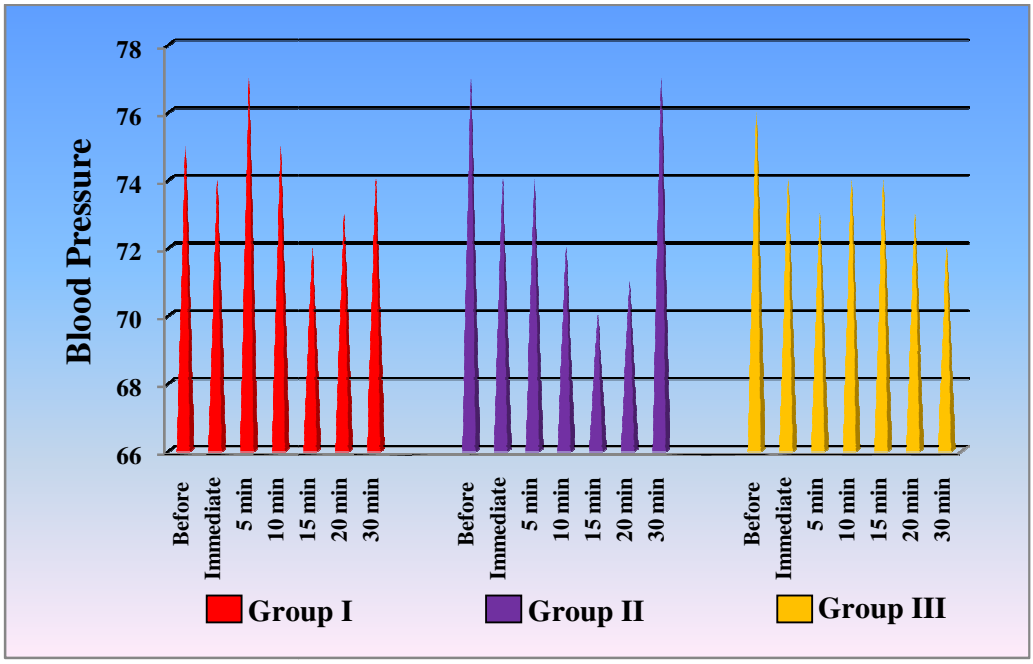
Graph-14: Comparison of diastolic blood pressure in Group-II (1:80000 dilution)



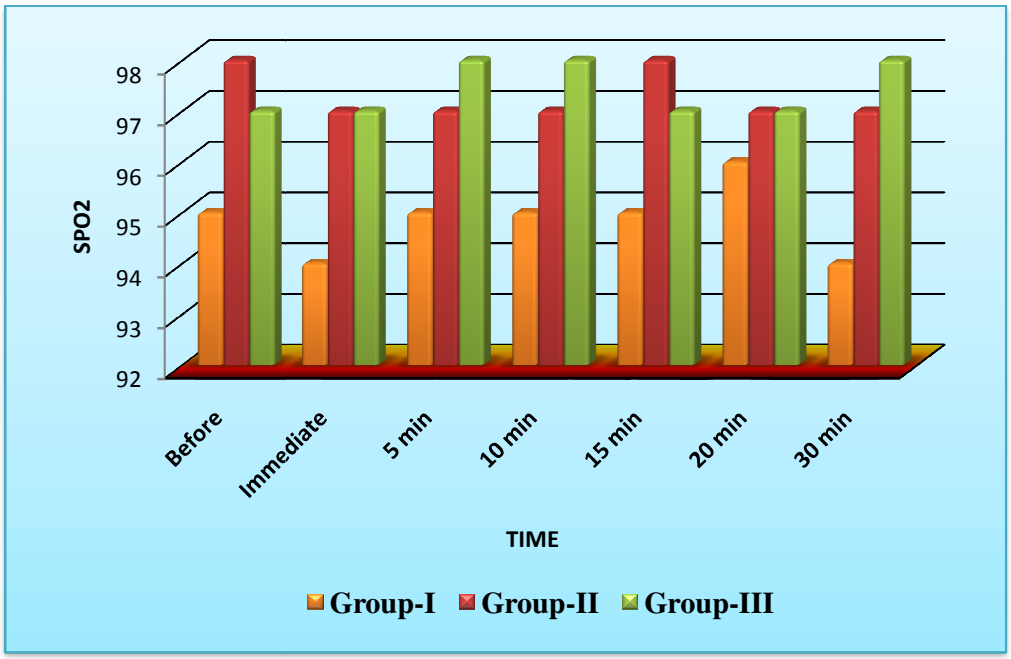
Graph-15: Comparison of diastolic blood pressure in Group-III (1:200000 dilution)



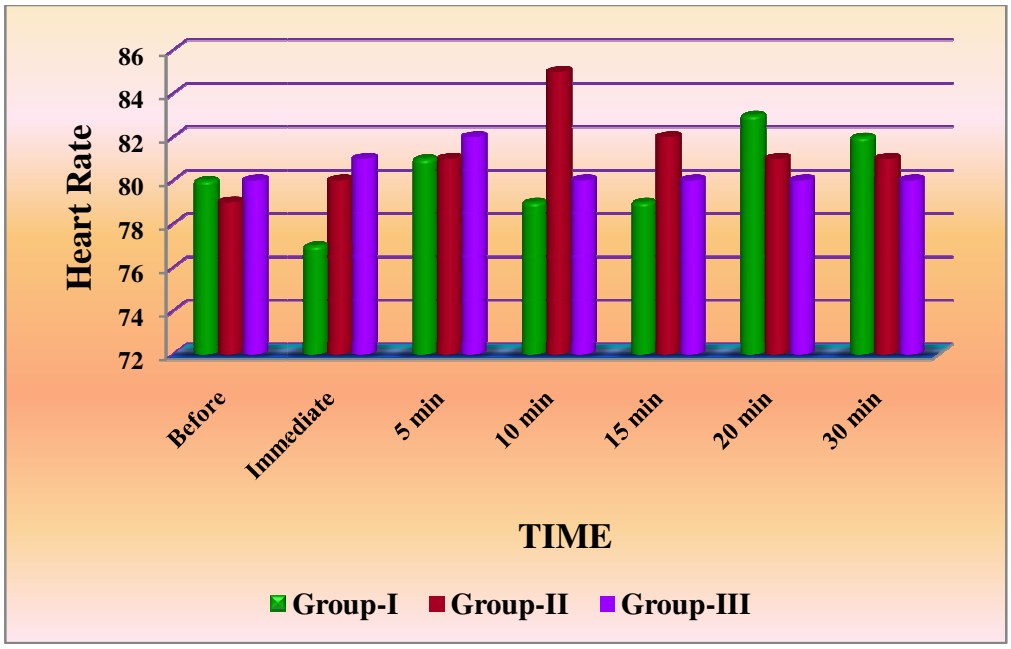
Graph-16: Multiple comparison of diastolic blood pressure with different time intervals within the groups



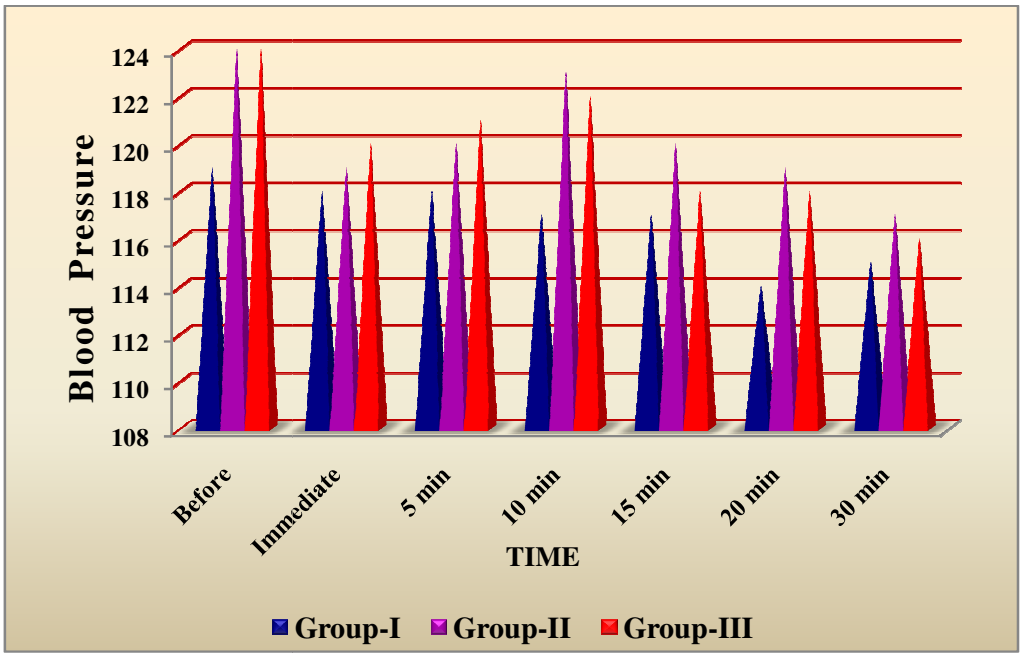
Graph-17 : Multiple comparison of mean values of SPO_2 of groups at different time period



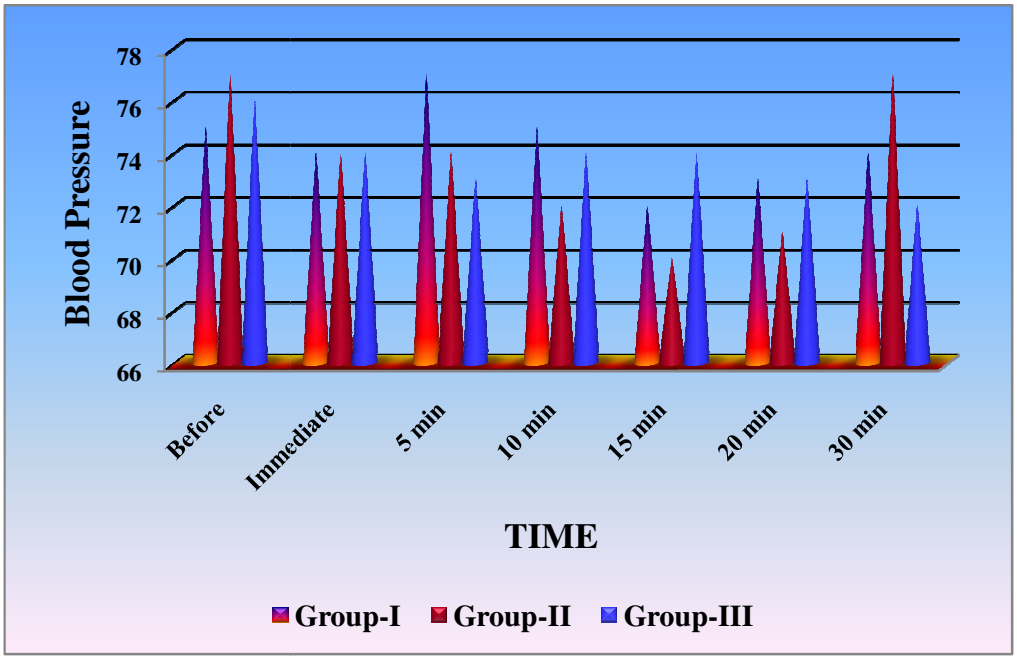
Graph-18: Multiple comparison of mean values of heart rate of groups at different time periods



Graph-19: Multiple comparison of mean values of systolic blood pressure of groups at different time periods



Graph-20: Multiple comparison of mean values of diastolic blood pressure of groups at different time periods



Statistical analysis within the group shows no significant changes in oxygen saturation in group I, group II and group III in any of the recorded time periods.

Group I showed only minor variations in HR which was not significant. Group II presented with a gradual increase in HR after LA administration with its peak increase at 10min of study period by 5 beats/min. Then it gradually decreased at 15 min and 20 min., reaching the pre administration levels at 30min. A mild increase in HR was noticed in group III by 5 min after administration, but was not significant. The increase in HR was significant in Group II compared with Group I and Group III.

The SBP in group I was seen to decrease after administration of LA, but was not statistically significant. In group II a decrease in SBP was noticed immediately after administration compared to pre administration values with a mean decrease of 5mmHg, and was then found to increase gradually at 5min and 10min. After 10min, SBP again decreased at 15min, 20min and 30min. time periods with a mean decrease of 7mm Hg from pre administration levels. Group III elicited a decrease in SBP showing a maximum decrease at 30 min with a mean of 6 mmHg. The fall in SBP was more in group II on comparing with group III, but was not significant.

Group I showed only minor changes in DBP without any statistical significance. In Group II there was a decrease in DBP after administration of LA with a maximum decrease seen at 15 min by 6 mmHg from pre administration levels. It then gradually increased over 20min and reaching the pre administration levels at 30min. Group III showed a gradual decrease in DBP with a maximum decrease seen at 30 min, with mean decrease being 5 mmHg.

Lignocaine with adrenaline combination is the most widely used local anaesthetic agent in dental practice[46]. The addition of adrenaline to local anaesthesia becomes essential in minor surgical procedures as adrenaline counteracts the known vasodilator activity of lignocaine by causing vasoconstriction and there by acting as a chemical tourniquet. This improves the depth and duration of anaesthesia and also reduces the systemic anaesthetic toxicity[52].

However these vasoconstrictors have been reported to influence blood pressure and heart rate[1,20]. Epinephrine stimulates adrenergic receptors which are responsible for their vasoconstrictor and other properties. There are two basic categories of adrenergic receptors, alpha adrenergic receptors and beta adrenergic receptors. Alpha adrenergic effects leads to peripheral vasoconstriction and beta adrenergic agonism causes increased rate and force of contraction of the heart and vasodilatation in muscles[23]. Thus epinephrine increases the systolic pressure and heart rate and decreases diastolic pressure. This property of epinephrine raises the controversy regarding the use of epinephrine in local anaesthetics in patients with cardiovascular disease. Though such effects of local anaesthesia with different epinephrine concentrations have been investigated, it is not well established. This study was designed to compare hemodynamic changes upon administration of local anaesthetic agents with their different concentrations of adrenaline through a period thirty minutes in otherwise healthy individuals.

Meechan et al[23] reported that cardiac transplant patients experienced a significant tachycardia 10 min after intra oral injection of the epinephrine-containing local anaesthetic solution with out any change in the systolic and diastolic blood

pressure. In the present study a similar increase in heart rate was noticed at 10 min period in lignocaine with adrenaline 1:80,000 concentration.

Another study by Meral et al[32] comparing the effects of lignocaine with or without adrenaline on hemodynamic changes during third molar surgery showed clinically negligible increase in HR and BP and oxygen saturation after injection of LA with epinephrine. Meechan and Rawlins in 1987 also found no significant changes in HR or SBP associated with the injection of 50 micrograms of adrenaline[53]. In 1988 they demonstrated a reduction in diastolic blood pressure following administration of adrenaline containing local anaesthetics[54]. The present study we found out no changes in oxygen saturation and a reduction in DBP at 15 min by 6mmHg from pre administration levels in lignocaine with adrenaline 1:80,000 concentration.

Chaudhry et al[47] observed a decrease of 21mm Hg in systolic blood pressure in hypertensive patients while administered with 3.6 ml of a 2% lignocaine with 1:100,000 epinephrine. A fall in DBP as well as HR was also noticed in this study. Gedik[31] and Takahashi et al[55] also noticed a decrease in SBP and HR during administration of local anaesthetic containing adrenaline in healthy patients. They observed a 4-5mmHg decrease in SBP after five minutes in a group that received 10micrograms of epinephrine in 2% lignocaine with a final volume of 4ml. We found a 7 mm Hg decrease in SBP after administration of 1.8 ml of lignocaine with adrenaline 1:80,000 concentration and 6 mm Hg decrease in SBP for lignocaine with adrenaline 1:2,00,000 in healthy individuals.

The decrease in BP may be attributed to very small plasma concentration of epinephrine in dental local anaesthesia [30]. The amount of epinephrine in two

cartridges of dental local anaesthetic injection is 45 micrograms. This concentration is reported to have predominant effect on cardiac ß-2 receptors and little effect on alpha receptors. When this is combined with lignocaine, which itself is a vasodilator, results in decrease in BP in otherwise healthy patients[30].

In another study conducted to record changes in hemodynamic parameters during functional endoscopic sinus surgery (FESS) after local infiltration with and without epinephrine showed a significant fall in blood pressure after injection of lignocaine with epinephrine (1:200,000) as compared to other groups that received a normal saline injection which was used as a placebo[35]. Hanuman et al[28] reported hemodynamic changes after scalp surgery using different concentrations of lignocaine and epinephrine. They concluded that lignocaine alone does not produce any significant change in hemodynamic parameters. This is in accordance with the present study which revealed insignificant haemodynamic changes associated with plain lignocaine.

Niwa et al[24] reported a 4.1% increase in SBP and 5.1% increase in HR immediately after injection of lignocaine with adrenaline. No change in diastolic blood pressure was noted. Knoll-Kohler et al[56] also demonstrated a slight increase in HR after injection of low dose epinephrine.

Clintron et al[57] reported that dental anaesthesia with 1.0ml of 2% lignocaine with 1:1,00,000 epinephrine caused no significant change in HR or blood pressure and was well tolerated by patients with recent myocardial infarction. Davenport[58] also reported insignificant hemodynamic changes with moderate doses of epinephrine used during periodontal surgery.

The amount of epinephrine absorbed following local dental anaesthesia has been hypothesized to be less than the amount produced endogenously in response to pain due to inadequate anaesthesia or due to anxiety associated with the procedure[59]. Studies investigating the administration of 1.8 ml of 2% lidocaine with epinephrine1:100,000 (18µg) reported elevated plasma epinephrine levels with minimal cardiovascular changes[11]. Troullos et al demonstrated elevated circulating levels of epinephrine following administration of 8 dental cartridges of lignocaine with adrenaline 1:1,00,000 and associated elevations in systolic pressure,heart rate, and myocardial oxygen consumption[12].The plasma epinephrine threshold for increase in heart rate is reported to be 50- 100 pg/ml[60]. The threshold level for increases in systolic blood pressure is 75 - 125 pg/ml, while 150 -200 pg/ml results in decreases in diastolic blood pressure [60].

Bader et al[29] reported that local anaesthesia with epinephrine used in hypertensive patients is associated with increase in SBP for 4 mmHg and no higher for normotensive subjects. HR was higher in patients receiving epinephrine than that with plain lignocaine group and DBP was found to decrease for both normotensive and hypertensive individuals. These changes were similar to the haemodynamic changes noted in the present study.

In this study, oxygen saturation did not show any significant changes before and after administration of local anaesthesia. This was in accordance with the results obtained by Meral[32] ,Vasconcellos[38] and Liau[37].

Considering hemodynamic study parameters, heart rate seen in group I(plain lignocaine) showed only minor variations which was not significant.In group II, there was an increase in the HR with its peak at 10min by 5 beats/min. Group III also

showed an increase in the heart rate but was not statistically significant. The rise in HR was in accordance with the studies by Niwa et al[24] and Knoll-Kohler [56].

The present study also showed that the pre administration SBP was higher than the values recorded at any other time periods. A decrease in systolic blood pressure was noted after administration in all the three groups compared to the pre administration levels. Group I elicited only a mild decrease in SBP. In group II there was a decrease noted immediately after administration with a mean decrease of 5mmHg followed by an elevation in SBP with its peak at 10 min period. It was again found to decrease at 15 min. 20 min and 30 min periods with a mean decrease of 7mmHg from pre administration levels. In Group III a decrease in SBP was noted at 30 min with a mean of 6 mmHg. Chaudhry et al[47] and Takahashi et al[55] also noticed similar haemodynamic changes while using local anaesthetics with adrenaline.

In this study, a decrease in the diastolic blood pressure was noted at 15 min and 20 min by 6 mmHg and then gradually increases, reaching the pre administration levels at 30 min. in group II. Group III showed a gradual decrease in DBP with a maximum decrease seen at 30 min, with mean decrease being 5 mmHg. Meechan[54] and Gedik[31] also demonstrated a reduction in diastolic blood pressure following administration of adrenaline containing local anaesthetics.

According to Hagigath[33] the increase in HR and SBP before administration can be attributed to needle phobia and stress during pre administration period .Once the needle is withdrawn, there is disappearance of fear and stress leading to a decrease in HR and SBP immediately after injection.

Dionne et al[12] reported that intraoral injections can cause endogenous epinephrine release which along with exogenously administered epinephrine causing a rise in HR and SBP during post injection period.

Pallasch et al[61] reported that epinephrine usually increases HR, SBP, myocardial oxygen consumption and cardiac automaticity but reduces DBP. This explains for the reduction in DBP in group II and group III following local anaesthesia administration in the present study.

In this study we found minor variations in HR, SBP and DBP with adrenaline concentrations of 1:80,000 and 1:2,00,000 dilutions when compared to plain lignocaine. All these variations were found to be more in lignocaine with adrenaline1:80,000 group (GroupII), even though it was not prominent.

The present study was conducted on a local population of sample size thirty evaluating their haemodynamic changes using pulse oxymetry while administering lignocaine alone and lignocaine with adrenaline in different concentration of 1:80,000 and 1: 2,00,000 in otherwise healthy individuals. It was noticed that oxygen saturation did not show any variations while using lignocaine with different concentrations of adrenaline. Those patients who received plain lignocaine 2% showed only minor variations in heart rate which was not significant after administration while patients who received lignocaine with adrenaline showed an increase in heart rate which was more in 1:80,000 group compared to 1:2,00,000 group. The decrease in systolic blood pressure was also found to be more in 1:80,000 group than 1:2,00,000 group. Diastolic blood pressure did not show any variations in those patients who received plain lignocaine but was noted to be decreasing in patients receiving local anaesthesia with adrenaline in concentration of 1:80,000. Adrenaline in 1:2,00,000 concentration also showed a decrease in diastolic blood pressure , but was not as low as that of adrenaline 1:80,000.

This analysis showed variation in heart rate, systolic blood pressure and diastolic blood pressure in patients who received lignocaine with adrenaline concentrations of 1:80,000 and 1:2,00,000. These variations were more in adrenaline concentrations of 1:80,000 than 1:2,00,000, but was not marked.

To conclude, even though use of lignocaine with adrenaline is associated with changes in blood pressure and heart rate, clinically they are less significant and such changes did not produce any marked systemic untoward outcome at any of the studied adrenaline concentrations of 1:80,000 or 1:2,00,000.

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